```
=> file biosis caba caplus embase japio lifesci medline scisearch
=> e sanders ira/au
E1            2     SANDERS ILSE M/AU
E2            1     SANDERS ILYSSA/AU
E3           82 --> SANDERS IRA/AU
E4            9     SANDERS IRA DR/AU
E5            2     SANDERS IRL R III/AU
E6            2     SANDERS IRWIN T/AU
E7           10     SANDERS IRYNA/AU
E8            2     SANDERS IRYNA F/AU
E9         1180     SANDERS J/AU
E10           1     SANDERS J 2ND/AU
E11         206     SANDERS J A/AU
E12           4     SANDERS J A C/AU

=> s e3-e4 and ((allergic rhinitis)or(allergic dermatitis))
L1            2 ("SANDERS IRA"/AU OR "SANDERS IRA DR"/AU) AND ((ALLERGIC RHINITI
                S) OR(ALLERGIC DERMATITIS))

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2            2 DUP REM L1 (0 DUPLICATES REMOVED)

=> d bib ab kwic 1-
YOU HAVE REQUESTED DATA FROM 2 ANSWERS - CONTINUE? Y/(N):y

L2   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2010 ACS on STN
AN   2004:467981  CAPLUS <<LOGINID::20100927>>
DN   141:17606
TI   Use of a clostridial neurotoxin for the treatment of mammalian
     physiological reaction of IgE antibodies present upon contact with the
     corresponding antigen
IN     ***Sanders, Ira***
PA   USA
SO   PCT Int. Appl., 28 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.        DATE
     ---------------     ----   --------    -------------------    --------
PI   WO 2004048519        A2    20040610    WO 2003-US37286        20031120
     WO 2004048519        A3    20040701
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM,
             PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN,
             TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
             ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK,
             TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG
     CA 2507115           A1    20040610    CA 2003-2507115        20031120
     AU 2003295769        A1    20040618    AU 2003-295769         20031120
     EP 1565210           A2    20050824    EP 2003-786972         20031120
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
```

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK
US 20060008462 A1 20060112 US 2005-535504 20050518
PRAI US 2002-427749P P 20021121
WO 2003-US37286 W 20031120
AB A method is disclosed for blocking or reducing physiol. reaction in a mammal to the interaction of IgE antibodies present in the mammal upon contact with the corresponding antigen, by the administration to the mammal of a therapeutically effective amt. of a neurotoxin derived from Clostridia sp.
RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
IN ***Sanders, Ira***
IT Allergy
(***allergic*** ***dermatitis*** ; clostridial neurotoxin for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)
IT Allergy
Inflammation
Nose, disease
(***allergic*** ***rhinitis*** ; clostridial neurotoxin for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

L2 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2002:10234 CAPLUS <<LOGINID::20100927>>
DN 136:64160
TI Methods for using tetanus toxin for beneficial purposes in animals (mammals)
IN ***Sanders, Ira***
PA USA
SO PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2002000172	A2	20020103	WO 2001-US20523	20010628
WO 2002000172	A3	20030807		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2413301	A1	20020103	CA 2001-2413301	20010628
AU 2001070219	A	20020108	AU 2001-70219	20010628
EP 1365800	A2	20031203	EP 2001-948786	20010628
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY, TR				
JP 2004521067	T	20040715	JP 2002-504955	20010628
US 20040248188	A1	20041209	US 2002-312705	20021224
US 7494661	B2	20090224		

```
     MX 2003000014        A     20040913    MX 2003-14             20030107
     IN 2003DN00052       A     20090320    IN 2003-DN52           20030115
     IN 239628            A1    20100402
     ZA 2003000701        A     20050509    ZA 2003-701            20030127
     AU 2007203272        A1    20070802    AU 2007-203272         20070713
     US 20090060953       A1    20090305    US 2008-288077         20081016
PRAI US 2000-214569P      P     20000628
     AU 2001-270219       A3    20010628
     AU 2001-70219        T0    20010628
     WO 2001-US20523      W     20010628
     US 2002-312705       A1    20021224
AB   Methods of using tetanus toxin to modulate or control neural functions or
     nonneural cellular activities at selected sites in animals, particularly
     in mammals, and more particularly in humans, are provided.  Pharmaceutical
     formulations to modulate neural functions or non-neural cellular
     activities of an animal at selected sites in animals, particularly in
     mammals, and more particularly in humans are also provided.  Uses of
     tetanus toxin in prepn. of medicaments for methods of treating clin.
     disorders or symptoms of animals, particularly mammals and more
     particularly humans are also provided.
OSC.G   6     THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)
RE.CNT  4     THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
              ALL CITATIONS AVAILABLE IN THE RE FORMAT
IN     ***Sanders, Ira***
IT   Allergy
        (  ***allergic***     ***dermatitis***  ; tetanus toxin for therapeutic
        use)


=> s ((allergic rhinitis)or(allergic dermatitis))
L3       63552 ((ALLERGIC RHINITIS) OR(ALLERGIC DERMATITIS))

=> s l3 and neurotoxin
L4         101 L3 AND NEUROTOXIN

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5           48 DUP REM L4 (53 DUPLICATES REMOVED)

=> d bib ab kwic 1-
YOU HAVE REQUESTED DATA FROM 48 ANSWERS - CONTINUE? Y/(N):y

L5   ANSWER 1 OF 48  LIFESCI    COPYRIGHT 2010 CSA on STN DUPLICATE 1
AN   2010:328356  LIFESCI <<LOGINID::20100927>>
TI   The roles of a Th2 cytokine and CC chemokine in children with stable
     asthma: Potential implication in eosinophil degranulation
AU   Kim, Chang K.; Kita, Hirohito; Callaway, Zak; Kim, Hyo B.; Choi, Jungi;
     Fujisawa, Takao; Shin, Bo M.; Koh, Young Y.
CS   1Pediatric Asthma and Allergy Center, Inje University Sanggye Paik
     Hospital, Seoul, Korea
SO   Pediatric Allergy and Immunology, (20100600) vol. 21, no. 4p2, pp.
     e697-e704.
     ISSN: 0905-6157.
DT   Journal
FS   F
LA   English
SL   English
```

AB Kim CK, Kita H, Callaway Z, Kim HB, Choi J, Fujisawa T, Shin BM, Koh YY. The roles of a Th2 cytokine and CC chemokine in children with stable asthma: Potential implication in eosinophil degranulation.
Pediatr Allergy Immunol 2010: 21: e697-e704.
[copy] 2010 John Wiley & Sons A-STh2 cytokine IL-5 and CC chemokine eotaxin are thought to be key regulators of eosinophils in bronchial asthma. However, their involvement in children with stable asthma (SA) has not been determined. We investigated the roles of IL-5 and eotaxin in eosinophil degranulation in children with SA. Induced sputum was obtained from 30 SA, 21 ***allergic*** ***rhinitis*** (AR), and 22 non-atopic healthy control (HC) children. We measured sputum levels of IL-5, eotaxin, and eosinophil indices [percentage eosinophils, eosinophil-derived ***neurotoxin*** (EDN), and eosinophil-cationic protein (ECP)]. We also examined correlations of IL-5 and eotaxin with eosinophil indices. Sputum percentage eosinophils and EDN and ECP levels were significantly higher in the SA group than in the HC group, while only the sputum EDN and ECP levels were significantly higher in the AR group than in the HC group. Unexpectedly, sputum levels of IL-5 were not significantly different among the three groups; however, the levels of eotaxin were higher in the SA group when compared to the HC group. No significant correlations were found between IL-5 and percentage eosinophils, EDN, or ECP levels; in contrast, eotaxin levels correlated significantly with percentage eosinophils (Rs = 0.638; p = 0.0001), EDN (Rs = 0.522; p = 0.003), and ECP levels (Rs = 0.630 and p = 0.0002). The elevated levels and good correlations of eotaxin with sputum eosinophil indices, and no elevation or correlation of IL-5 with these indices, suggest that CC chemokine eotaxin may play a more important role in eosinophil degranulation in children with SA.

AB . . . roles of IL-5 and eotaxin in eosinophil degranulation in children with SA. Induced sputum was obtained from 30 SA, 21 ***allergic*** ***rhinitis*** (AR), and 22 non-atopic healthy control (HC) children. We measured sputum levels of IL-5, eotaxin, and eosinophil indices [percentage eosinophils, eosinophil-derived ***neurotoxin*** (EDN), and eosinophil-cationic protein (ECP)]. We also examined correlations of IL-5 and eotaxin with eosinophil indices. Sputum percentage eosinophils and. . .

UT ***Allergic*** ***rhinitis*** ; Asthma; CC chemokines; Children; Cytokines; Degranulation; Eosinophil-derived ***neurotoxin*** ; Helper cells; Hypersensitivity; Interleukin 5; Leukocytes (eosinophilic); Lymphocytes T; Sputum; eotaxin

L5 ANSWER 2 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2009:539230 CAPLUS <<LOGINID::20100927>>
DN 150:512868
TI The Human Eosinophil Proteome. Changes Induced by Birch Pollen Allergy
AU Woschnagg, Charlotte; Forsberg, Jens; Engstroem, Ake; Odreman, Federico; Venge, Per; Garcia, Rodolfo C.
CS Department of Medical Sciences, Clinical Chemistry, Uppsala University, Uppsala, 75185, Swed.
SO Journal of Proteome Research (2009), 8(6), 2720-2732
CODEN: JPROBS; ISSN: 1535-3893
PB American Chemical Society
DT Journal
LA English
AB Proteins from human eosinophils were sepd. bidimensionally and identified by mass spectrometry (336 spots/bands, 98 different proteins). Of these,

24.7% belonged to the cytoskeleton/migration group. Highly basic proteins (11.3%) were concd. in the granule-contg. cell fraction. We detected novel hyperacidic forms of cofilin-1, profilin-1 and adenylyl cyclase-assocd. protein, and hyperbasic forms of eosinophil-derived ***neurotoxin*** /eosinophil protein X and major basic protein homolog. We also found evidence of the triglycosylation of the heavy chain of eosinophil peroxidase. In addn., through comparative 2D image anal., spot quantification and MS, it was found that hsc70, actin-capping protein and hyperacidic forms of eosinophil peroxidase heavy chain are overexpressed in cells from birch pollen allergic subjects, at the peak of a season. The link between these findings and an increased cellular antigen-presenting capacity and motility are discussed.

RE.CNT 72 THERE ARE 72 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . granule-contg. cell fraction. We detected novel hyperacidic forms of cofilin-1, profilin-1 and adenylyl cyclase-assocd. protein, and hyperbasic forms of eosinophil-derived ***neurotoxin*** /eosinophil protein X and major basic protein homolog. We also found evidence of the triglycosylation of the heavy chain of eosinophil. . .

IT Cytokines
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(EDN (eosinophil-derived ***neurotoxin***); changes of human eosinophil proteome induced by birch pollen allergy)

IT ***Allergic*** ***rhinitis***
Antigen presentation
Betula
Eosinophil
Human
Pollen
Protein microarray technology
Proteomics
(changes of human eosinophil proteome induced by birch pollen allergy)

L5 ANSWER 3 OF 48 SCISEARCH COPYRIGHT (c) 2010 The Thomson Corporation on STN

AN 2009:480251 SCISEARCH <<LOGINID::20100927>>

GA The Genuine Article (R) Number: 431JW

TI Toll-like receptor 2 is important for the T(H)1 response to cutaneous sensitization

AU Geha, Raif (Reprint)
CS Childrens Hosp, Div Immunol, Room 10210, Karp Family Res Bldg, 1 Blackfan Circle, Boston, MA 02115 USA (Reprint)
E-mail: raif.geha@childrens.harvard.edu
AU Jin, Haoli; Kumar, Lalit; Mathias, Clinton; Oettgen, Hans; Geha, Raif (Reprint)
CS Childrens Hosp, Div Immunol, Boston, MA 02115 USA
E-mail: raif.geha@childrens.harvard.edu
AU Jin, Haoli; Kumar, Lalit; Mathias, Clinton; Oettgen, Hans; Geha, Raif (Reprint)
CS Harvard Univ, Sch Med, Dept Pediat, Boston, MA 02115 USA
E-mail: raif.geha@childrens.harvard.edu
AU Zurakowski, David
CS Childrens Hosp, Dept Anesthesia & Surg, Boston, MA 02115 USA
AU Gorelik, Leonid
CS Biogen Idec Inc, Cambridge, MA USA
CYA USA

SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (APR 2009) Vol. 123, No. 4,

pp. 875-882.
ISSN: 0091-6749.
PB MOSBY-ELSEVIER, 360 PARK AVENUE SOUTH, NEW YORK, NY 10010-1710 USA.
DT Article; Journal
LA English
REC Reference Count: 44
ED Entered STN: 23 Apr 2009
Last Updated on STN: 19 Mar 2010
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Background: Atopic dermatitis and allergic contact dermatitis are skin disorders triggered by epicutaneous sensitization with protein antigens and contact sensitization with haptens, respectively. Skin is colonized with bacteria, which are a source of Toll-like receptor (TLR) 2 ligands.

Objective: We sought to examine the role of TLR2 in marine models of atopic dermatitis and allergic contact dermatitis.

Methods: TLR2(-/-) mice and wild-type littermates were epicutaneously sensitized with ovalbumin (OVA) or contact sensitized with oxazolone (OX). Skin histology was assessed by means of hematoxylin and eosin staining and immunohistochemistry. Ear swelling was measured with a micrometer. Cytokine mRNA expression was examined by means of quantitative RT-PCR. Antibody levels and splenocyte secretion of cytokines in response to OVA stimulation were measured by means of ELISA. Dendritic cells were examined for their ability to polarize T-cell receptor/OVA transgenic naive T cells to T(H)1 and T(H)2.

Results: In response to OVA sensitization, TLR2(-/-) mice experienced skin infiltration with eosinophils and CD4(+) cells, as well as upregulation of T(H)2 cytokine mRNAs that was comparable with that seen in wild-type littermates. In contrast, epidermal thickening, IFN-gamma expression in the skin, IFN-gamma production by splenocytes, and IgG2a anti-OVA antibody levels were impaired in TLR2(-/-) mice. After OX ear challenge, contact sensitized TLR2(-/-) mice exhibited defective ear swelling with impaired cellular infiltration, decreased epidermal thickening and local IFN-gamma expression, and impaired OX-specific IgG2a responses. Dendritic cells from TLR2(-/-) mice induced significantly lower production of IFN-gamma but normal IL-4 and IL-13 production in naive T cells.

Conclusions: These results indicate that TLR2 promotes the IFN-gamma response to cutaneously introduced antigens. (J Allergy Clin Immunol 2009;123:875-82.)
STP KeyWords Plus (R): EOSINOPHIL-DERIVED ***NEUROTOXIN*** ; HUMAN DENDRITIC CELLS; ATOPIC-DERMATITIS; T-CELLS; CYTOKINE/CHEMOKINE PRODUCTION; ***ALLERGIC*** ***DERMATITIS*** ; IMMUNE-RESPONSES; INTERFERON-GAMMA; INNATE IMMUNITY; CUTTING EDGE

L5 ANSWER 4 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2009014016 EMBASE <<LOGINID::20100927>>
TI Increased production of cysteinyl leukotrienes and prostaglandin D2 during human anaphylaxis.
AU Ono, E. (correspondence); Taniguchi, M.; Mita, H.; Fukutomi, Y.; Higashi, N.; Akiyama, K.
CS Clinical Research Center for Allergy and Rheumatology, National Hospital Organization, Sagamihara National Hospital, 18-1 Sakuradai, Sagamihara, Kanagawa 228-8522, Japan. e-ono@sagamihara-hosp.gr.jp
AU Ono, E. (correspondence); Miyazaki, E.; Kumamoto, T.
CS Third Department of Internal Medicine, Oita University Faculty of Medicine, Yuhu, Oita, Japan. e-ono@sagamihara-hosp.gr.jp

SO Clinical and Experimental Allergy, (January 2009) Vol. 39, No. 1, pp. 72-80.
Refs: 28
ISSN: 0954-7894; E-ISSN: 1365-2222 CODEN: CLEAEN
PB Blackwell Publishing Ltd, 9600 Garsington Road, Oxford, OX4 2XG, United Kingdom.
CY United Kingdom
DT Journal; Article
FS 006 Internal Medicine
005 General Pathology and Pathological Anatomy
015 Chest Diseases, Thoracic Surgery and Tuberculosis
026 Immunology, Serology and Transplantation
029 Clinical and Experimental Biochemistry
LA English
SL English
ED Entered STN: 3 Feb 2009
Last Updated on STN: 3 Feb 2009
AB Background: Anaphylaxis is a life-threatening syndrome resulting from the sudden release of mast cell- and basophil-derived mediators into the circulation. However, pathological evidence of the association between inflammatory mediators and human anaphylaxis is insufficient. Objective: The aim of this study was to better understand the relationship between in vivo production of inflammatory mediators and the pathogenesis of anaphylaxis. We also sought to evaluate mast cell activation in anaphylaxis. Methods: We measured the concentrations of various inflammatory mediators in urine samples, which were collected from 32 anaphylactic patients during the onset of anaphlaxis and during clinical remission, 21 patients with asthma on acute exacerbation and 15 healthy control subjects. Blood and urine specimens were collected from the patients after provocation test. Urinary leukotriene E4 (LTE4), 9.alpha., 11.beta.-prostaglandin F2 (9.alpha., 11.beta.-PGF2), eosinophil-derived ***neurotoxin*** (EDN) and leukotriene B4 glucuronide (LTBG) concentrations were determined by enzyme immunoassay, and the activity of plasma platelet-activating factor acetylhydrolase and serum tryptase concentration were measured using commercially available kits. Results: Significantly higher concentrations of urinary LTE4 and 9.alpha., 11.beta.-PGF2, which immediately decreased during clinical remission, were observed in the anaphylactic patients than in asthmatic patients on acute exacerbation and healthy control subjects. Concentrations of EDN and LTBG were not significantly different among the anaphylactic patients, asthmatic patients on acute exacerbation and healthy subjects. There was a significant correlation between urinary LTE4 and 9.alpha., 11.beta.-PGF2 concentrations in the anaphylactic patients (r=0.672, P=0.005, n=32). In addition, LTE4 concentration in patients with anaphylactic shock is significantly elevated compared with that in patients without anaphylactic shock. Conclusions: This is a report on the significant increase in urinary LTE4 and 9.alpha., 11.beta.-PGF2 concentrations during anaphylaxis. Urinary LTE4 and 9.alpha., 11.beta.-PGF2 concentrations may be a reliable marker of endogenous production of inflammatory mediators associated with anaphylaxis. .COPYRGT. 2008 The Authors.
AB . . . urine specimens were collected from the patients after provocation test. Urinary leukotriene E4 (LTE4), 9.alpha., 11.beta.-prostaglandin F2 (9.alpha., 11.beta.-PGF2), eosinophil-derived ***neurotoxin*** (EDN) and leukotriene B4 glucuronide (LTBG) concentrations were determined by enzyme immunoassay, and the activity of plasma platelet-activating factor acetylhydrolase. . .
CT Medical Descriptors:

adult
allergic rhinitis
anaphylactic shock
*anaphylaxis: ET, etiology
article
asthma
atopic dermatitis
basophil
blood sampling
*cell activation
clinical article
clinical assessment
clinical feature
control group
controlled study
correlation analysis
cytokine production
disease association
disease exacerbation
drug hypersensitivity
emergency ward
enzyme blood level
enzyme. . .

L5 ANSWER 5 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN
AN 2010086272 EMBASE <<LOGINID::20100927>>
TI [Eosinophilic esophagitis in children and adults: A new diagnostic and management challenge of the XXI century].
Eozynofilowe zapalenie przelyku u dzieci i doroslych - Nowe wyzwanie diagnostyczno-terapeutyczne XXI wieku.
AU Iwanczak, Barbara, Dr. Prof. (correspondence)
CS Katedra i Klinika Pediatrii, Gastroenterologii i Zywienia AM, ul. Sklodowskiej-Curie 50/52, 50-369 Wroclaw. barbara@iwanczak.com
SO Family Medicine and Primary Care Review, (July-September 2008) Vol. 10, No. 3, pp. 853-860.
Refs: 39
ISSN: 1734-3402
PB Wydawnictwo Continuo, ul.lelewela 4, Wroclaw woj.dolnoslaskie, 53505, Poland.
CY Poland
DT Journal; General Review; (Review)
FS 005 General Pathology and Pathological Anatomy
011 Otorhinolaryngology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
048 Gastroenterology
LA Polish
SL English; Polish
ED Entered STN: 2 Mar 2010
Last Updated on STN: 2 Mar 2010
AB Eosinophilic esophagitis is a disease characterized by eosinophilic infiltration of the esophageal mucosa and clinical symptoms such as dysphagia, food impaction, symptoms of gastroesophageal reflux disease resistant to pharmacological treatment. In endoscopic examination rings, longitudinal furrows, white coating and stenosis of the esophagus are visible. Infiltration of more than 15 eosinophils in high power field,

hypertrophy of basal and papillary layers are observed in histopathologic examination. Differential diagnostics should exclude: gastroesophageal reflux disease, Crohns' disease, eosinophilic syndrome, mucosal infections, and inflammatory diseases of connective tissue. In pathogenesis a connection with pollen and food allergy, bronchial asthma, ***allergic*** ***rhinitis*** and atopic dermatitis should be taken into consideration. Fifty fold increase in eotaxin-3, increase in eosinophilic ***neurotoxin*** , IL-5 and IL-13 were observed in patients with eosinophilic esophagitis. Elimination diet, local and systemic use of glucosteroids, leukotriene inhibitors, anti-IL-5 antibodies and proton pump inhibitors in case of acidic reflux as well as dilatation of the esophagus are applied in the treatment of the disease.

AB . . . syndrome, mucosal infections, and inflammatory diseases of connective tissue. In pathogenesis a connection with pollen and food allergy, bronchial asthma, ***allergic*** ***rhinitis*** and atopic dermatitis should be taken into consideration. Fifty fold increase in eotaxin-3, increase in eosinophilic ***neurotoxin*** , IL-5 and IL-13 were observed in patients with eosinophilic esophagitis. Elimination diet, local and systemic use of glucosteroids, leukotriene inhibitors,. . .

CT Medical Descriptors:
adult
allergic rhinitis
asthma
atopic dermatitis
child
clinical feature
Crohn disease
diagnostic approach route
diet restriction
differential diagnosis
disease association
disease resistance
dysphagia
endoscopy
*eosinophilic esophagitis: DI, diagnosis
*eosinophilic esophagitis: DT, drug therapy
esophagus dilatation
food allergy
gastroesophageal reflux
histopathology
human
mucosa. . .
drug therapy
interleukin 13: EC, endogenous compound
interleukin 5: EC, endogenous compound
*interleukin 5 antibody: DT, drug therapy
*leukotriene receptor blocking agent: DT, drug therapy
neurotoxin: EC, endogenous compound
*proton pump inhibitor: DT, drug therapy

RN (interleukin 13) 148157-34-0; (***neurotoxin***) 39386-17-9

L5 ANSWER 6 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 2

AN 2008:664278 BIOSIS <<LOGINID::20100927>>

DN PREV200800664277

TI Hepatocyte Growth Factor Suppresses Production of Reactive Oxygen Species and Release of Eosinophil-Derived ***Neurotoxin*** from Human Eosinophils.

AU Ito, Wataru; Takeda, Masahide; Fujita, Miyoshi; Kamada, Yumiko; Kato, Hikari; Chiba, Takahito; Yamaguchi, Kazutoshi; Ueki, Shigeharu; Kayaba, Hiroyuki; Kanehiro, Arihiko; Chihara, Junichi [Reprint Author]

CS Akita Univ, Sch Med, Dept Clin and Lab Med, 1-1-1 Hondo, Akita 0108543, Japan
chihara@hos.akita-u.ac.jp

SO International Archives of Allergy and Immunology, (2008) Vol. 147, No. 4, pp. 331-337.
CODEN: IAAIEG. ISSN: 1018-2438.

DT Article

LA English

ED Entered STN: 27 Nov 2008
Last Updated on STN: 3 Dec 2008

AB Background: Reactive oxygen species (ROS) and eosinophilic granule proteins such as eosinophil-derived ***neurotoxin*** (EDN) are known to damage bronchial tissue and cause airway hyperresponsiveness (AHR) in asthma. Hepatocyte growth factor (HGF) regulates various biological activities and is known to be a multifunctional factor. In our previous study, we found that HGF suppressed allergic airway inflammation and AHR in a murine model of asthma. However, there have been few reports regarding the detailed mechanism of the anti-allergic effect of HGF in asthma. In this study, we investigated the potential of recombinant HGF to regulate the production of ROS and the release of EDN from human eosinophils. Methods: Eosinophils were isolated from subjects with mild eosinophilia by modified CD16-negative selection. We investigated the expression of CD69, an activation marker of eosinophils, on eosinophils, using flow cytometry. Further, ROS production from eosinophils was analyzed using luminol-dependent chemiluminescence, and EDN release was measured by ELISA. Results: Treatment with HGF suppressed interleukin-5-induced upregulation of CD69 expression, ROS production and EDN release from human eosinophils. Conclusion: Taken together, these data suggest that in asthma, HGF attenuates allergic airway inflammation and AHR through at least the suppression of ROS production and EDN release from eosinophils.

TI Hepatocyte Growth Factor Suppresses Production of Reactive Oxygen Species and Release of Eosinophil-Derived ***Neurotoxin*** from Human Eosinophils.

AB Background: Reactive oxygen species (ROS) and eosinophilic granule proteins such as eosinophil-derived ***neurotoxin*** (EDN) are known to damage bronchial tissue and cause airway hyperresponsiveness (AHR) in asthma. Hepatocyte growth factor (HGF) regulates various. . .

IT . . .

IT Diseases
eosinophilia: blood and lymphatic disease
Eosinophilia (MeSH)

IT Diseases
asthma: respiratory system disease, immune system disease
Asthma (MeSH)

IT Diseases
allergic ***rhinitis*** : respiratory system disease, immune system disease
Rhinitis, Allergic, Perennial (MeSH)

IT Diseases
allergic airway disease: respiratory system disease, immune system

disease
IT Chemicals & Biochemicals
CD69: expression; interleukin-5; recombinant hepatocyte growth factor [rHGF]; reactive oxygen species [ROS]: production; eosinophil-derived ***neurotoxin*** [EDN]

L5 ANSWER 7 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2007:845505 CAPLUS <<LOGINID::20100927>>
DN 147:197194
TI Pharmaceutical composition containing several botulic toxins
IN Berruet, Laure
PA Societe de Conseils de Recherches et d'Applications Scientifiques (S.C.R.A.S.), Fr.
SO PCT Int. Appl., 15pp.
CODEN: PIXXD2
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2007085728	A2	20070802	WO 2007-FR134	20070124
	WO 2007085728	A3	20071115		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
	RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
	FR 2896693	A1	20070803	FR 2006-732	20060127
	FR 2896693	B1	20080314		
	AU 2007209213	A1	20070802	AU 2007-209213	20070124
	AU 2007209213	A2	20090108		
	CA 2640342	A1	20070802	CA 2007-2640342	20070124
	EP 1981530	A2	20081022	EP 2007-730852	20070124
	EP 1981530	B1	20090708		
	R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
	AT 435658	T	20090715	AT 2007-730852	20070124
	JP 2009526759	T	20090723	JP 2008-551821	20070124
	CN 101360509	A	20090204	CN 2007-80001572	20080612
	IN 2008DN05595	A	20080926	IN 2008-DN5595	20080627
	MX 2008009212	A	20080730	MX 2008-9212	20080717
	KR 2008086522	A	20080925	KR 2008-7018358	20080725
	US 20080292612	A1	20081127	US 2008-180725	20080728
PRAI	FR 2006-732	A	20060127		
	WO 2007-FR134	W	20070124		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The invention concerns a compn. comprising at least: one botulic ***neurotoxin*** type A1, and one botulic ***neurotoxin*** type A the amino acid sequence of which has at least 5% difference with the amino acid sequence of the botulic ***neurotoxin*** type A1. A

pharmaceutical injection contained botulic ***neurotoxin*** type A1 250 units, and botulic ***neurotoxin*** type A2 from Clostridium botulinum 250 units. Efficacy of the compn. in the treatment of a patient suffering from blepharospasm is shown.

OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

AB The invention concerns a compn. comprising at least: one botulic ***neurotoxin*** type A1, and one botulic ***neurotoxin*** type A the amino acid sequence of which has at least 5% difference with the amino acid sequence of the botulic ***neurotoxin*** type A1. A pharmaceutical injection contained botulic ***neurotoxin*** type A1 250 units, and botulic ***neurotoxin*** type A2 from Clostridium botulinum 250 units. Efficacy of the compn. in the treatment of a patient suffering from blepharospasm. . .

IT Acne
Allergic ***rhinitis***
Analgesics
Arthritis
Clostridium botulinum
Constipation
Cosmetics and personal care products
Dyskinesia
Dystonia
Endometriosis
Eye disease
Fibromyalgia
Human
Myalgia
Nervous system disease
Neuromuscular disease
Obesity
Osteoarthritis
Pharmaceutical injections
Psoriasis
Rheumatoid arthritis
Rhinitis
Surfactants
Vasoconstrictors
Wrinkle-preventing cosmetics
(pharmaceutical compn. contg. several botulic toxins)

L5 ANSWER 8 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2007:1449166 CAPLUS <<LOGINID::20100927>>
DN 148:45872
TI Simultaneous, separate, or sustained-release therapeutic use of a botulinum ***neurotoxin*** and an opiate compound
IN Auguet, Michel Didier; Favre, Christine; Chabrier de Lassauniere, Pierre Etienne
PA Societe de Conseils de Recherches et d'Applications Scientifiques Scras, Fr.
SO Fr. Demande, 23pp.
CODEN: FRXXBL
DT Patent
LA French
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	FR 2902341	A1	20071221	FR 2006-5368	20060616

AU 2007259122 A1 20071221 AU 2007-259122 20070611
CA 2655488 A1 20071221 CA 2007-2655488 20070611
WO 2007144493 A2 20071221 WO 2007-FR956 20070611
WO 2007144493 A3 20080417
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA
EP 2037956 A2 20090325 EP 2007-788861 20070611
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, PL, PT, RO, SE, SI, SK, TR, AL, BA, HR, MK, RS
JP 2009539948 T 20091119 JP 2009-514842 20070611
MX 2008015254 A 20081217 MX 2008-15254 20081128
CN 101466401 A 20090624 CN 2007-80022016 20081212
US 20090232851 A1 20090917 US 2008-305191 20081216
PRAI FR 2006-5368 A 20060616
WO 2007-FR956 W 20070611
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The invention discloses a compn. including at least one botulinum ***neurotoxin*** and at least an opiate deriv. or salt thereof. The invention also discloses a product of at least one botulinum ***neurotoxin*** and at least one opiate deriv., or salt thereof, as a combination product for, simultaneous, sep. or sustained-release therapeutic use in the treatment or the prevention of pain and neuromuscular disorders.
RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Simultaneous, separate, or sustained-release therapeutic use of a botulinum ***neurotoxin*** and an opiate compound
AB The invention discloses a compn. including at least one botulinum ***neurotoxin*** and at least an opiate deriv. or salt thereof. The invention also discloses a product of at least one botulinum ***neurotoxin*** and at least one opiate deriv., or salt thereof, as a combination product for, simultaneous, sep. or sustained-release therapeutic use. . .
ST pain analgesic botulinum ***neurotoxin*** opiate combination; neuromuscular disorder treatment botulinum ***neurotoxin*** opiate combination
IT Muscle disease
((spasmodic) torticolis; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Polysaccharides
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(2-Hydroxyethyl amidon; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Biliary tract
(Oddi's sphincter, dysfunction; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Digestive tract disease
(achalasia; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Pain
(adiposis dolorosa; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Disease, animal
(algoneurodystrophy; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Surgery
(amputation; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Anus
(anal fissure; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Surfactants
(anionic; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Disease, animal
(anismus; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Prostate gland disease
(benign hyperplasia; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT ***Allergic*** ***rhinitis***
Alopecia
Analgesics
Anti-inflammatory agents
Antiarthritics
Antidepressants
Antimigraine agents
Antiobesity agents
Antirheumatic agents
Arthritis
Bone disease
Bone fracture
Central nervous system agents
Constipation
Depression
Dyskinesia
Dystonia
Endocrine system disease
Endometriosis
Eye disease
Fibromyalgia
Gastrointestinal agents
Gout
Headache
Human
Joint disease
Laxatives
Mental and behavioral disorders
Muscle disease
Myalgia
Nervous system agents
Nervous system disease
Neuromuscular disease

Obesity
Pain
Pancreatitis
Poisoning, biological
Prophylaxis
Psoriasis
Psychotropics
Rheumatoid arthritis
Rhinitis
Surfactants
Urinary system disease
Wrinkle-preventing cosmetics
(botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Opioids
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Polysaccharides
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Surfactants
(cationic; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Stroke
(central pain from; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Paralysis
(cerebral; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Disease, animal
(chronic, pain assocd.with; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Drug delivery systems
(coated; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Skin
(cosmetic blemish; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Hip
(coxarthrosis; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Nerve, disease
(diabetic neuropathy; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Joint, anatomical
(elbow, epicondylitis; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Eye
(eye contour wrinkle; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Head and Neck
(face, eyebrow furrows; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Head and Neck

(face, facial asymmetry; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Head and Neck
(face, wrinkle; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Skin
(glabellar wrinkle; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Pain
(hyperalgesia; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Sweat
(hyperhidrosis and bromhidrosis; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Eye disease
(hyperlacrimation; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Bladder disease
(incontinence; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Pain
(inflammatory pain; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Eye
(lid, blepharospasm; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Headache
(migraine; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Muscle disease
(muscle spasm, spasmodic torticolis; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Muscle disease
(muscle spasm; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Nerve, disease
Pain
(neuralgia, post-herpetic; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Pain
(neuropathic pain; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Toxins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(neurotoxins; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Surfactants
(nonionic; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Drug toxicity
(pain assocd. with antitumor agent use; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Antitumor agents
(pain assocd. with use of; botulinum ***neurotoxin*** -opiate compd.

combination for treatment of pain or neuromuscular disorder)
IT AIDS (disease)
Burn
Multiple sclerosis
(pain assocd. with; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Neoplasm
(pain assocd.with; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Herpesviridae
Human herpesvirus
(post-herpetic neuralgia; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Surgery
(pre- and post-operative pain; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Stomach disease
(pyloric valve spasm; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Nervous system disease
(radiculopathy, pain assocd.with; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Pain
(regional complex pain syndrome; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Disease, animal
(sciatica; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Muscle disease
(shoulder rotator cuff muscle disorder; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Muscle relaxants
(smooth; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Bladder disease
(spasm; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Muscle relaxants
(spasmolytics; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Bladder disease
(spastic bladder; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Neuromuscular disease
(spasticity; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Vision disorders
(strabismus; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Drug interactions
(synergistic; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Tendon
(tear; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)
IT Thalamus

(thalamic lesion, pain assocd. with; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Injury
(trauma; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Bladder disease
(urinary retention; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT Skin
(wrinkle; botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT 57-27-2D, Morphine, analogs and derivs., biological studies 57-42-1D, Pethidine, analogs and derivs. 64-31-3D, Morphine sulfate, analogs and derivs. 71-68-1D, Hydromorphone hydrochloride, analogs and derivs. 76-42-6D, Oxycodone, analogs and derivs. 76-57-3D, Codeine, analogs and derivs. 125-28-0D, Dihydrocodeine, analogs and derivs. 125-29-1D, Hydrocodone, analogs and derivs. 437-38-7D, Fentanyl, analogs and derivs. 466-99-9D, Hydromorphone, analogs and derivs. 469-62-5D, Dextropropoxyphene, analogs and derivs. 20594-83-6D, Nalbuphine, analogs and derivs. 27203-92-5D, Tramadol, analogs and derivs. 52485-79-7D, Buprenorphine, analogs and derivs. 56030-54-7D, Sufentanil, analogs and derivs. 71195-58-9D, Alfentanil, analogs and derivs. 93384-43-1, Dysport 93384-44-2, Botulin B 93384-45-3, Botulin C 93384-46-4, Botulin D 93384-47-5, Botulin E 107231-12-9, Botulin 107231-13-0, Botulin C1 107231-15-2, Botulin F 107231-16-3, Botulin G 132875-61-7D, Remifentanyl, analogs and derivs. 863191-48-4, Botulin A 1 1112056-09-3, Botulin A2
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

IT 34344-66-6, Polysorbic acid
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(botulinum ***neurotoxin*** -opiate compd. combination for treatment of pain or neuromuscular disorder)

L5 ANSWER 9 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
DUPLICATE 3
AN 2007:397894 BIOSIS <<LOGINID::20100927>>
DN PREV200700394007
TI A dual antagonist for chemokine CCR3 receptor and histamine H-1 receptor.
AU Suzuki, Keiko [Reprint Author]; Morokata, Tatsuaki; Morihira, Koichiro; Sato, Ippei; Takizawa, Satoko; Kaneko, Masayuki; Takahashi, Koichiro; Shimizu, Yasuaki
CS Astellas Pharma Inc, Drug Discovery Res, Pharmacol Res Labs, Dept Immunol,Yodogawa Ku, 1-6,Kashima 2 Chome, Osaka 5328514, Japan
keiko-suzuki@jp.astellas.com
SO European Journal of Pharmacology, (JUN 1 2007) Vol. 563, No. 1-3, pp. 224-232.
CODEN: EJPHAZ. ISSN: 0014-2999.
DT Article
LA English
ED Entered STN: 18 Jul 2007
Last Updated on STN: 18 Jul 2007
AB Eosinophilic chemokines and histamine play distinct but important roles in allergic diseases. Inhibition of both eosinophilic chemokines and

histamine, therefore, is an ideal strategy for the treatment of allergic inflammation, such as asthma, ***allergic*** ***rhinitis*** , and atopic dermatitis. YM-344484 was found to potently inhibit both the CCL11-induced Ca2+ influx in human CCR3-expressing cells (K-b=1.8 nM) and histamine-induced Ca2+ influx in histamine H-1 receptor-expressing PC3 cells (K-b=47 nM). YM-344484 also inhibited the CCL11-induced chemotaxis of human CCR3-expressing cells (IC50=6.2 nM) and CCL11-induced eosinophil-derived ***neurotoxin*** release from human eosinophils (IC50=19 nM). Orally administered YM-344484 inhibited the increase in histamine-induced vascular permeability in mice (82% inhibition at a dose of 10 mg/kg) and the accumulation of eosinophils in a mouse asthma model (74% at a dose of 300 mg/kg). These results indicate that YM-344484, a novel and functional dual antagonist for chemokine CCR3 receptor and histamine H, receptor, is an attractive candidate for development as a novel anti-allergic inflammation drug, (c) 2007 Elsevier B.V. All rights reserved.

AB. . . of both eosinophilic chemokines and histamine, therefore, is an ideal strategy for the treatment of allergic inflammation, such as asthma, ***allergic*** ***rhinitis*** , and atopic dermatitis. YM-344484 was found to potently inhibit both the CCL11-induced Ca2+ influx in human CCR3-expressing cells (K-b=1.8 nM). . . receptor-expressing PC3 cells (K-b=47 nM). YM-344484 also inhibited the CCL11-induced chemotaxis of human CCR3-expressing cells (IC50=6.2 nM) and CCL11-induced eosinophil-derived ***neurotoxin*** release from human eosinophils (IC50=19 nM). Orally administered YM-344484 inhibited the increase in histamine-induced vascular permeability in mice (82% inhibition. . .

IT . . . of Organisms
eosinophil: immune system, blood and lymphatics

IT Diseases
asthma: respiratory system disease, immune system disease
Asthma (MeSH)

IT Diseases
allergic ***rhinitis*** : respiratory system disease, immune system disease
Rhinitis, Allergic, Perennial (MeSH)

IT Diseases
atopic dermatitis: integumentary system disease, genetic disease, immune system. . .

L5 ANSWER 10 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2007:260413 CAPLUS <<LOGINID::20100927>>
DN 147:268109
TI Expanding clinical uses of botulinum neurotoxins
AU Moore, A. Peter
CS The Walton Centre for Neurology and Neurosurgery, Liverpool University, Liverpool, UK
SO Treatments from Toxins (2007), 163-194. Editor(s): Foster, Keith A.; Hambleton, Peter; Shone, Clifford C. Publisher: CRC Press LLC, Boca Raton, Fla.
CODEN: 69IYSV; ISBN: 978-0-8493-2709-4
DT Conference; General Review
LA English
AB A review on clin. uses of botulinum neurotoxins. The development and assessment of new indications for botulinum neurotoxins are discussed. These indications include glandular hypersecretion, hypersalivation, focal

hyperhidrosis (excessive sweating), rhinorrhea (runny nose) and ***allergic*** ***rhinitis*** , bladder hyperreflexia, detrusor sphincter dyssynergia, urethrospasm, prostate disorders, esophageal spasm, obesity, anal fissure, pelvic floor dysfunction, rectal pain, and pain.

OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

RE.CNT 104 THERE ARE 104 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . indications for botulinum neurotoxins are discussed. These indications include glandular hypersecretion, hypersalivation, focal hyperhidrosis (excessive sweating), rhinorrhea (runny nose) and ***allergic*** ***rhinitis*** , bladder hyperreflexia, detrusor sphincter dyssynergia, urethrospasm, prostate disorders, esophageal spasm, obesity, anal fissure, pelvic floor dysfunction, rectal pain, and pain.

ST review botulinum ***neurotoxin*** glandular hypersecretion bladder hyperreflexia esophageal spasm

IT ***Allergic*** ***rhinitis***
(botulinum neurotoxins showed therapeutic efficacy in treatment of ***allergic*** ***rhinitis*** in patient)

L5 ANSWER 11 OF 48 SCISEARCH COPYRIGHT (c) 2010 The Thomson Corporation on STN

AN 2007:981201 SCISEARCH <<LOGINID::20100927>>

GA The Genuine Article (R) Number: 208KA

TI Botulinum toxin therapy in the ovalbumin-sensitized rat

AU Hou, Yi-Ping (Reprint)

CS Lanzhou Univ, Dept Anat, Neurobiol Lab, Sch Basic Med Sci, 199 Donggang Xi Rd, Lanzhou 730000, Peoples R China (Reprint)

AU Wen, Wei-Dong; Yuan, Fang; Wang, Jian-Lin

CS Lanzhou Univ, Dept Anat, Neurobiol Lab, Sch Basic Med Sci, Lanzhou 730000, Peoples R China; Lanzhou Univ, Sch Life Sci, Lanzhou 730000, Peoples R China; NW Univ Natl, Fac Med, Lanzhou, Peoples R China
E-mail: houyiping@lzu.edu.cn

CYA Peoples R China

SO NEUROIMMUNOMODULATION, (2007) Vol. 14, No. 2, pp. 78-83.
ISSN: 1021-7401.

PB KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.

DT Article; Journal

LA English

REC Reference Count: 28

ED Entered STN: 4 Oct 2007
Last Updated on STN: 4 Oct 2007

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Objective: The aim of this study was to determine whether intranasal administration of botulinum toxin type A (BTX-A) could relieve the typical symptoms of ***allergic*** ***rhinitis*** (AR) and alter substance P (SP)- and vasoactive intestinal peptide (VIP)- immunoreactive (IR) expression in nasal mucosa of AR animals sensitized with ovalbumin (OVA). Methods: AR was induced by intraperitoneal injection of OVA followed by its repeated intranasal instillation in female Wistar rats. Some AR animals were intranasally treated with a cotton strip containing BTX-A (10 U per nostril) for 1 h. After BTX-A treatment, OVA was repeatedly instilled in AR and AR + BTX-A groups every 2 days for 10 days. Subsequently, nasal symptoms were evaluated, and nasal secretions collected. Finally, the nasal mucosae of all animals were prepared for histological and immunohistochemical assessment. Results: BTX-A administration alleviated typical AR symptoms including rhinorrhea, nasal itching and sneezing, and subsequent intranasal repeated challenge with

OVA did not trigger AR symptoms. After BTX-A treatment, inflammatory histological characteristics within the nasal mucosa of AR animals were absent, but atrophy of serous glands was observed. BTX-A decreased dense SP-IR and VIP-IR cells and fibers within and beneath the epithelium, around blood vessels and close to serous glands in AR animals. Conclusion: Local BTX-A treatment is an effective method to reduce AR symptoms. BTX-A decreased the excessive SP-IR and VIP-IR expression induced by OVA. Therefore, BTX-A may affect the nasal mucosa via the suppression of neuropeptides, playing a major role in autonomous mucosal innervation in the pathophysiology of AR. Copyright c 2007 S. Karger AG, Basel.

AB . . . this study was to determine whether intranasal administration of botulinum toxin type A (BTX-A) could relieve the typical symptoms of ***allergic*** ***rhinitis*** (AR) and alter substance P (SP)- and vasoactive intestinal peptide (VIP)- immunoreactive (IR) expression in nasal mucosa of AR. . .

ST Author Keywords: ***allergic*** ***rhinitis*** ; botulinum toxin type A; ovalbumin; substance P; vasoactive intestinal peptide

STP KeyWords Plus (R): EXPERIMENTAL ***ALLERGIC*** ***RHINITIS*** ; ***NEUROTOXIN*** -A; PROTEIN; INFLAMMATION; NEURONS; AIRWAYS; MUCOSA

L5 ANSWER 12 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN

AN 2006:542307 CAPLUS <<LOGINID::20100927>>

DN 145:50777

TI Fusion proteins comprising non-cytotoxic protease, translocation, protease cleavage site, and targeting moieties for the treatment of diseases

IN Foster, Keith; Chaddock, John; Marks, Philip; Stancombe, Patrick; Durose, Lyndsey

PA Health Protection Agency, UK

SO PCT Int. Appl., 114 pp.
CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2006059113	A2	20060608	WO 2005-GB4606	20051201
WO 2006059113	A3	20060928		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2005311106	A1	20060608	AU 2005-311106	20051201
CA 2593707	A1	20060608	CA 2005-2593707	20051201
CN 101098885	A	20080102	CN 2005-80046021	20051201
EP 1874807	A2	20080109	EP 2005-818352	20051201
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
JP 2008521428	T	20080626	JP 2007-543914	20051201

	BR 2005015760	A	20080729	BR 2005-15760	20051201
	IN 2007DN04183	A	20070831	IN 2007-DN4183	20070601
	US 20080064092	A1	20080313	US 2007-853517	20070911
	US 20090004174	A1	20090101	US 2007-792076	20071119
PRAI	GB 2004-26397	A	20041201		
	WO 2005-GB4606	W	20051201		
	US 2007-792076	A1	20071119		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention provides a single-chain, polypeptide fusion protein comprising: (1) a non-cytotoxic protease, or a fragment thereof, capable of cleaving a protein of the exocytic fusion app. of a target cell; (2) a Targeting Moiety that is capable of binding to a Binding Site on the target cell, which Binding Site is capable of undergoing endocytosis to be incorporated into an endosome within the target cell; (3) a protease cleavage site at which site the fusion protein is cleavable by a protease, such that the protease cleavage site is located between the non-cytotoxic protease and the Targeting Moiety; (4) and a translocation domain that is capable of translocating the protease or protease fragment from within an endosome, across the endosomal membrane and into the cytosol of the target cell. Specifically, the non-cytotoxic protease is a clostridial ***neurotoxin*** , and in particular the light or L-chains of Clostridium botulinum ***neurotoxin*** (botulin serotypes B and C), which are capable of cleaving the SNARE proteins (synaptobrevin, syntaxin, or SNAP-25) of the exocytic fusion app. Translocation moieties comprise a portion or fragment of the heavy H-chain of the clostridial ***neurotoxin*** approx. equiv. to the N-terminal half of the H-chain. Preferred Targeting Moieties may comprise proteinase-activated receptor ligands (e.g., PAR1), parathyroid hormone, vasoactive intestinal polypeptide (VIP), gastrin-releasing peptide, the Arg-Gly-Asp (RGD) peptide motif, linear and cyclic THALWHT peptides, and atrial natriuretic peptide (ANP). Proteolytic cleavage of the fusion proteins vis the protease cleavage site yields di-chain polypeptide medicaments for treating, preventing, or ameliorating a medical condition.

OSC.G 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . endosome, across the endosomal membrane and into the cytosol of the target cell. Specifically, the non-cytotoxic protease is a clostridial ***neurotoxin*** , and in particular the light or L-chains of Clostridium botulinum ***neurotoxin*** (botulin serotypes B and C), which are capable of cleaving the SNARE proteins (synaptobrevin, syntaxin, or SNAP-25) of the exocytic fusion app. Translocation moieties comprise a portion or fragment of the heavy H-chain of the clostridial ***neurotoxin*** approx. equiv. to the N-terminal half of the H-chain. Preferred Targeting Moieties may comprise proteinase-activated receptor ligands (e.g., PAR1), parathyroid. . .

ST noncytotoxic protease translocation targeting cleavage fusion protein; ***neurotoxin*** translocation targeting cleavage moiety fusion protein; botulin translocation targeting cleavage moiety fusion protein; sequence fusion protein botulin targeting cleavage moiety

IT Allergy
Inflammation
Nose, disease
(***allergic*** ***rhinitis*** , treatment of; fusion proteins comprising non-cytotoxic protease, translocation, protease cleavage

site, and targeting moieties for the treatment of diseases)

L5 ANSWER 13 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2006:374234 CAPLUS <<LOGINID::20100927>>
DN 144:404402
TI Low-adenosine antisense oligonucleotides for treatment of airway disorders associated with bronchoconstriction, lung inflammation, allergy and surfactant depletion
IN Nyce, Jonathan W.; Metzger, W. James
PA East Carolina University, USA
SO U.S., 485 pp., Cont.-in-part of U.S. Ser. No. 543,679.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 8

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 7034007	B1	20060425	US 2003-543679	20030101
	US 5994315	A	19991130	US 1995-474497	19950607
	US 6040296	A	20000321	US 1995-472527	19950607
	US 6025339	A	20000215	US 1996-757024	19961126
	US 20030087845	A1	20030508	US 1998-93972	19980609
	US 6825174	B2	20041130		
	AU 9918574	A	19990506	AU 1999-18574	19990303
	AU 724817	B2	20000928		
	AU 2002050710	A	20020808	AU 2002-50710	20020628
	US 20050014711	A1	20050120	US 2004-758451	20040114
PRAI	US 1995-472527	A2	19950607		
	US 1995-474497	A2	19950607		
	US 1996-757024	A2	19961126		
	US 1997-59160P	P	19970917		
	US 1998-16464	B2	19980130		
	US 1998-93972	A2	19980609		
	US 1999-127958P	P	19990406		
	US 2000-543679	A2	20000404		
	AU 1996-60295	A3	19960603		
	AU 2000-71749	A3	20001122		

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB An in vivo method of selectively delivering a nucleic acid to a target gene or mRNA, comprises the topical administration, e.g. to the respiratory system, of a subject of a therapeutic amt. of an oligonucleotide (oligo) that is antisense to a mRNA complementary to the gene in an amt. effective to reach the target polynucleotide and reducing or inhibiting expression. The compn. and formulations are used for prophylactic, preventive and therapeutic treatment of ailments assocd. with impaired respiration, lung allergy(ies) and/or inflammation and depletion lung surfactant or surfactant hypoprodn., such as pulmonary vasoconstriction, inflammation, allergies, ***allergic*** ***rhinitis*** , asthma, impeded respiration, lung pain, cystic fibrosis, bronchoconstriction. The antisense oligos are designed to alleviate hyper-responsiveness to adenosine or increased levels of adenosine, wherein the antisense oligo is 7-60 nucleotides long and contains .ltoreq.15% adenosine. The treatment of this invention may be administered directly as an aerosol into the respiratory system of a subject so that the agent has direct access to the lungs, in an amt. effective to reduce or inhibit the symptoms of the ailment. Thus, the

phosphorothioated oligo 5'-gatggaggggggcatggcggg-3' antisense to human adenosine A1 receptor mRNA, and related oligos specific for different regions of the A1 receptor mRNA for antisense to A2b and A3 receptors, are highly effective at countering or reducing effects mediated by the receptors they are targeted to. The activity of the antisense oligos is specific to the target and substitutively fails to inhibit another target, and results in extremely low or non-existent deleterious side effects or toxicity.

OSC.G 3 THERE ARE 3 CAPLUS RECORDS THAT CITE THIS RECORD (3 CITINGS)

RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . with impaired respiration, lung allergy(ies) and/or inflammation and depletion lung surfactant or surfactant hypoprodn., such as pulmonary vasoconstriction, inflammation, allergies, ***allergic*** ***rhinitis*** , asthma, impeded respiration, lung pain, cystic fibrosis, bronchoconstriction. The antisense oligos are designed to alleviate hyper-responsiveness to adenosine or increased. . .

IT Cytokines
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(EDN (eosinophil-derived ***neurotoxin***); low-adenosine antisense oligonucleotides for treatment of airway disorders assocd. with bronchoconstriction, lung inflammation, allergy and surfactant depletion)

L5 ANSWER 14 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN DUPLICATE 4

AN 2006445849 EMBASE <<LOGINID::20100927>>

TI [Is the seric eosinophil cationic protein level a valuable tool of diagnosis in clinical practice?].
Le dosage de la proteine cationique des eosinophiles est-il un marqueur utile pour l'interniste ?.

AU Moneret-Vautrin, D.-A. (correspondence)
CS Service de medecine interne, immunologie clinique et allergologie, hopital universitaire, CHU, 29, ave. Marechal-Lattre-de-Tassigny, 54035 Nancy cedex, France. a.moneret-vautrin@chu-nancy.fr

SO Revue de Medecine Interne, (Sep 2006) Vol. 27, No. 9, pp. 679-683.
Refs: 50
ISSN: 0248-8663; E-ISSN: 1768-3122 CODEN: RMEIDE
PUI S 0248-8663(06)00099-3
CY France
DT Journal; (Short Survey)
FS 015 Chest Diseases, Thoracic Surgery and Tuberculosis
017 Public Health, Social Medicine and Epidemiology
026 Immunology, Serology and Transplantation
048 Gastroenterology
006 Internal Medicine
LA French
SL French; English
ED Entered STN: 28 Sep 2006
Last Updated on STN: 28 Sep 2006

AB Scope: The eosinophil cationic protein (ECP) is one of the mediators released during eosinophil activation. These cells are effector cells taking part into the Th2-lymphocyte dependent allergic inflammation. Assaying ECP concentrations in blood and sputum may be useful in evaluating allergic inflammation (asthma and rhinitis). This summary considers the value of measuring ECP levels for the diagnosis of various

diseases where an eosinophil-mediated tissue inflammation plays a role. Current situation and salient points: Levels of eosinophil cationic protein have been determined in nasal secretions, sputum, gastric secretions, feces and serum. They are increased during seasonal ***allergic*** ***rhinitis*** and perennial rhinitis, allergic asthma and atopic dermatitis. They are also increased in various gastro-intestinal disorders, some of which are associated with IgE: eosinophil intestinal diseases (esophagitis, gastro-enteritis and colitis), gastro-intestinal food allergy and intestinal parasitoses. Finally, they are increased in non IgE-dependent disorders: non allergic asthma with aspirin intolerance, respiratory infections, sinonasal polyposis, Churg-Strauss disease and idiopathic hyper-eosinophilia (HES) syndrome. Perspectives: Assaying serum ECP could help in the diagnosis of several diseases. With parasitic disease the pathogenic progression may be accurately assessed, when serological tests are less indicative. ECP assay may point to non allergic asthma, either Fernand-Widal syndrome or Churg-Strauss disease. As for gastro-intestinal disorders, it indicates an eosinophilic tissue reaction. In the event of isolated hypereosinophilia, ECP assay may clarify whether it is benign or tending towards idiopathic HES. The assay of peroxidase and eosinophil-derived ***neurotoxin*** (EDN) should be also considered.

AB . . . eosinophil cationic protein have been determined in nasal secretions, sputum, gastric secretions, feces and serum. They are increased during seasonal ***allergic*** ***rhinitis*** and perennial rhinitis, allergic asthma and atopic dermatitis. They are also increased in various gastro-intestinal disorders, some of which are. . . hypereosinophilia, ECP assay may clarify whether it is benign or tending towards idiopathic HES. The assay of peroxidase and eosinophil-derived ***neurotoxin*** (EDN) should be also considered.

L5 ANSWER 15 OF 48 EMBASE DUPLICATE 5
AN 2006587044 EMBASE <<LOGINID::20100927>>
TI Botulinum toxin in primary care medicine.
AU Felber, Eric S., Dr. (correspondence)
CS Frankford Hospitals-Jefferson Health System, Philadelphia, PA, United States. efelbs@hotmail.com
AU Felber, Eric S., Dr. (correspondence)
CS 402 Chestnut Ct, Bensalem, PA 19020-4315, United States. efelbs@hotmail.com
SO Journal of the American Osteopathic Association, (Oct 2006) Vol. 106, No. 10, pp. 609-614.
Refs: 33
ISSN: 0098-6151; E-ISSN: 0098-6151 CODEN: JAOAAZ
CY United States
DT Journal; General Review; (Review)
FS 017 Public Health, Social Medicine and Epidemiology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
039 Pharmacy
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 15 Dec 2006
Last Updated on STN: 15 Dec 2006

AB Clostridium botulinum, a gram-positive anaerobic bacterium, produces a potent ***neurotoxin*** that causes muscle paralysis. The therapeutic use of botulinum toxin was discovered in the 1970s and has since been used to treat patients with a broad range of medical complaints. Botulinum toxin (BTX) is used in the primary care setting to treat conditions such as ***allergic*** ***rhinitis*** , hyperhidrosis, lichen simplex chronicus, migraine, myofascial pain syndrome, and certain task-specific idiopathic focal dystonias (eg, writer's cramp) - in addition to its more publicized use for cosmetic enhancement of the face. The expanding range of therapeutic applications for BTX make it necessary for primary care physicians to understand the biochemistry, preparation, indications, and interactions of BTX.

AB Clostridium botulinum, a gram-positive anaerobic bacterium, produces a potent ***neurotoxin*** that causes muscle paralysis. The therapeutic use of botulinum toxin was discovered in the 1970s and has since been used. . . broad range of medical complaints. Botulinum toxin (BTX) is used in the primary care setting to treat conditions such as ***allergic*** ***rhinitis*** , hyperhidrosis, lichen simplex chronicus, migraine, myofascial pain syndrome, and certain task-specific idiopathic focal dystonias (eg, writer's cramp) - in addition. . .

CT Medical Descriptors:
allergic rhinitis: DT, drug therapy
dose response
drug blood level
drug half life
drug mechanism
dystonia: DT, drug therapy
human
hyperhidrosis: DT, drug therapy
idiopathic disease
migraine: DT, drug therapy
muscle. . .

L5 ANSWER 16 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN

AN 2006254969 EMBASE <<LOGINID::20100927>>

TI Modulation of eosinophil functions by nitric oxide: Cyclic GMP-dependent and -independent mechanisms.

AU Ferreira, Heloisa H.A.
CS Laboratory of Inflammation Research, Sao Francisco University, Braganca Paulista, SP, Brazil.
AU Conran, Nicola; Antunes, Edson (correspondence)
CS Department of Pharmacology and Hemotherapy Center, Faculty of Medical Sciences, UNICAMP, P.O. Box 6111, 13084-971 Campinas, SP, Brazil. edson.antunes@uol.com.br
AU Antunes, Edson (correspondence)
CS Department of Pharmacology, Faculty of Medical Sciences, UNICAMP, P.O. Box 6111, 13084-971 Campinas, SP, Brazil. edson.antunes@uol.com.br

SO Anti-Inflammatory and Anti-Allergy Agents in Medicinal Chemistry, (Feb 2006) Vol. 5, No. 1, pp. 45-57.
Refs: 211
ISSN: 1871-5230
CY Netherlands
DT Journal; General Review; (Review)
FS 015 Chest Diseases, Thoracic Surgery and Tuberculosis
026 Immunology, Serology and Transplantation
029 Clinical and Experimental Biochemistry

030 Clinical and Experimental Pharmacology
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
LA English
SL English
ED Entered STN: 15 Jun 2006
Last Updated on STN: 15 Jun 2006
AB Recruitment of eosinophils into tissues is a feature of a variety of allergic diseases, including asthma and nasal allergy. Eosinophils secrete several preformed granule proteins (eosinophil peroxidase, major basic protein, eosinophil cationic protein and eosinophil-derived ***neurotoxin***) and newly-generated substances (oxygen-derived toxic metabolites, lipid mediators, cytokines and chemokines), which may contribute to the exacerbation of the allergic diseases. In the past decade, NO has been recognized as a major immunomodulatory mediator of inflammatory responses, particularly in the lung, where it is believed to play a pivotal role in modulating pulmonary eosinophilia and airways hyperresponsiveness in both allergic animals and humans, as evidenced by functional, biochemical and immunohistochemical studies. The NO-cGMP signaling cascade was initially implicated in the modulation of eosinophil functions; however, additional studies have demonstrated that direct cGMP-independent mechanisms may also play important roles in eosinophil functions. Much progress in understanding the influence of NO on eosinophil functions has been achieved with the use of selective and non-selective NOS inhibitors, as well as NO-donor compounds, along with NOS isoform gene knock-out mice. However, these studies have resulted in numerous controversies and conflicting findings, possibly as a consequence of the diversity of experimental models used, animal species employed, methods of immunization and challenge with allergens, amongst others. The present review summarizes the role of NO in modulating, in vivo and in vitro, eosinophil adhesion, chemotaxis, airways hyperresponsiveness and apoptosis, outlining the conflicting findings in the literature, with emphasis on the allergic inflammatory responses. .COPYRGT. 2006 Bentham Science Publishers Ltd.
AB . . . asthma and nasal allergy. Eosinophils secrete several preformed granule proteins (eosinophil peroxidase, major basic protein, eosinophil cationic protein and eosinophil-derived ***neurotoxin***) and newly-generated substances (oxygen-derived toxic metabolites, lipid mediators, cytokines and chemokines), which may contribute to the exacerbation of the allergic. . .
CT Medical Descriptors:
*allergic reaction: DT, drug therapy
*allergic reaction: ET, etiology
allergic rhinitis: DT, drug therapy
allergic rhinitis: ET, etiology
apoptosis
asthma: DT, drug therapy
asthma: ET, etiology
cell interaction
cell secretion
disease exacerbation
drug mechanism
enzyme activity
enzyme inhibition
*eosinophil
human
immunomodulation

in vitro study
in vivo study
leukocyte adherence
*leukocyte function
Loeffler. . .

L5 ANSWER 17 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2004:467981 CAPLUS <<LOGINID::20100927>>
DN 141:17606
TI Use of a clostridial ***neurotoxin*** for the treatment of mammalian physiological reaction of IgE antibodies present upon contact with the corresponding antigen
IN Sanders, Ira
PA USA
SO PCT Int. Appl., 28 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2004048519	A2	20040610	WO 2003-US37286	20031120
WO 2004048519	A3	20040701		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2507115	A1	20040610	CA 2003-2507115	20031120
AU 2003295769	A1	20040618	AU 2003-295769	20031120
EP 1565210	A2	20050824	EP 2003-786972	20031120
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
US 20060008462	A1	20060112	US 2005-535504	20050518
PRAI US 2002-427749P	P	20021121		
WO 2003-US37286	W	20031120		

AB A method is disclosed for blocking or reducing physiol. reaction in a mammal to the interaction of IgE antibodies present in the mammal upon contact with the corresponding antigen, by the administration to the mammal of a therapeutically effective amt. of a ***neurotoxin*** derived from Clostridia sp.
RE.CNT 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Use of a clostridial ***neurotoxin*** for the treatment of mammalian physiological reaction of IgE antibodies present upon contact with the corresponding antigen
AB . . . mammal upon contact with the corresponding antigen, by the administration to the mammal of a therapeutically effective amt. of a ***neurotoxin*** derived from Clostridia sp.
ST clostridial ***neurotoxin*** IgE antibody antigen reaction allergy treatment
IT Antibodies and Immunoglobulins

RL: BSU (Biological study, unclassified); BIOL (Biological study)
(IgE; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Medical goods
(adsorbent pledgets; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Allergy
(***allergic*** ***dermatitis*** ; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Allergy
Inflammation
Nose, disease
(***allergic*** ***rhinitis*** ; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Dermatitis
(allergic; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Bronchi, disease
Inflammation
(bronchitis; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Bronchi
(bronchoconstriction; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Drug delivery systems
(carriers, biodegradable; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Allergy
Allergy inhibitors
Antiasthmatics
Antitussives
Bronchodilators
Clostridium
Clostridium baratii
Clostridium botulinum
Clostridium butyricum
Clostridium tetani
Cough
Emphysema
Food allergy
Human
Lung, disease
Pruritus
(clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Allergens
Antigens
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Mucus
(excessive secretion related to allergic reaction; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Asthma
(hyperreactive; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Drug delivery systems
(inhalants; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Drug delivery systems
(injections; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Mucous membrane
(mucosal edema; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Drug delivery systems
(myringotoomy; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Drug delivery systems
(nasal; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Toxins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(neurotoxins; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Ear, disease
Inflammation
(otitis media, serous; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Inflammation
Nose, disease
(rhinitis, infectious; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Inflammation
Respiratory system, disease
(sinusitis; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Breathing (animal)
(sneezing; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT Drug delivery systems

(suppositories; clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

IT 93384-43-1, Botulin A 93384-44-2, Botulin B 93384-46-4, Botulin D 93384-47-5, Botulin E 107231-13-0, Botulin C1 107231-15-2, Botulin F 107231-16-3, Botulin G
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(clostridial ***neurotoxin*** for treatment of physiol. reaction of IgE antibodies present upon contact with corresponding antigen)

L5 ANSWER 18 OF 48 SCISEARCH COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2004:840897 SCISEARCH <<LOGINID::20100927>>
GA The Genuine Article (R) Number: 853AX
TI Peripheral blood eosinophils from patients with allergic asthma contain increased intracellular eosinophil-derived ***neurotoxin***
AU Sedgwick J B (Reprint)
CS Univ Wisconsin, Dept Med, CSC-3244, H6-355 Allergy, 600 Highland Ave, Madison, WI 53792 USA (Reprint)
AU Vrtis R F; Jansen K J; Kita H; Bartemes K; Busse W W
CS Univ Wisconsin, Dept Med, Madison, WI 53792 USA; Mayo Clin & Mayo Fdn, Rochester, MN 55905 USA
E-mail: jxs@medicine.wisc.edu
CYA USA
SO JOURNAL OF ALLERGY AND CLINICAL IMMUNOLOGY, (SEP 2004) Vol. 114, No. 3, pp. 568-574.
ISSN: 0091-6749.
PB MOSBY, INC, 11830 WESTLINE INDUSTRIAL DR, ST LOUIS, MO 63146-3318 USA.
DT Article; Journal
LA English
REC Reference Count: 25
ED Entered STN: 15 Oct 2004
Last Updated on STN: 15 Oct 2004
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Background: One mechanism of the eosinophil's contribution to airway inflammation in asthma is through release of cationic granule proteins to cause airway injury. Differences in either the intracellular concentration of granule proteins or the extent of activated degranulation between eosinophils from healthy patients and those with allergy and asthma could, therefore, relate to fundamental differences in this cell's function.

Objective: To identify phenotypic differences in eosinophil-derived ***neurotoxin*** (EDN) content and release in eosinophils from healthy patients, those with allergy, and those with allergy and asthma.

Methods: Peripheral blood eosinophils were isolated by negative anti-CD16 selection. Total intracellular and cytokine-activated release of EDN protein was measured by radioimmunoassay. EDN mRNA was assessed by real-time PCR.

Results: Eosinophils from patients with asthma contained significantly more EDN per cell than comparable cells from healthy patients, those with allergy but without asthma, or those with asthma treated with inhaled corticosteroids, but they had concentrations similar to airway eosinophils isolated from bronchoalveolar lavage fluid 48 hours after segmental bronchoprovocation with allergen. Furthermore, this increased granule protein was reflected in more EDN degranulation by IL-5- or GM-CSF-activated eosinophils when calculated as nanograms of protein

secreted but not when calculated as a percentage of total EDN release. Levels of EDN mRNA were similar in all subject groups.

Conclusions: These data suggest that peripheral blood eosinophils from subjects with untreated asthma have increased inflammatory capacity, as reflected by greater intracellular concentrations of EDN.

TI Peripheral blood eosinophils from patients with allergic asthma contain increased intracellular eosinophil-derived ***neurotoxin***

AB . . . allergy and asthma could, therefore, relate to fundamental differences in this cell's function.

Objective: To identify phenotypic differences in eosinophil-derived ***neurotoxin*** (EDN) content and release in eosinophils from healthy patients, those with allergy, and those with allergy and asthma.

Methods: Peripheral. . .

ST Author Keywords: eosinophil; eosinophil-derived neurotosin; degranulation; asthma; ***allergic*** ***rhinitis*** ; corticosteroids; hypereosinophilic syndrome

L5 ANSWER 19 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 6

AN 2005:107998 BIOSIS <<LOGINID::20100927>>

DN PREV200500107882

TI Concentrations of eosinophil-derived ***neurotoxin*** in the blood and urine of patients with allergic diseases.

AU Morioka, Junichiro; Tomita, Miyuki; Yoshizawa, Yoshitomo; Inamura, Hiroaki; Kurosawa, Motohiro [Reprint Author]

CS Gunma Inst Allergy and Asthma, Shin Ohra Hosp, 3233-1 Shinozuka, Takasaki, Gumma, 3700615, Japan
motohiro@kl.wind.ne.jp

SO Allergology International, (December 2004) Vol. 53, No. 4, pp. 359-367. print.
ISSN: 1323-8930 (ISSN print).

DT Article

LA English

ED Entered STN: 16 Mar 2005
Last Updated on STN: 16 Mar 2005

AB Background: It has been reported that measurements of eosinophil-derived ***neurotoxin*** (EDN) may be useful to determine eosinophil activities in allergic diseases. Methods: Serum, plasma and urine concentrations of EDN in patients with allergic diseases were measured by enzyme-linked immunosorbent assay. Samples of blood and urine were obtained from the same patients when their symptoms were active and when patients were well. The number of eosinophils in peripheral blood was also counted. Results: The median concentrations of EDN in blood and urine and the number of eosinophils in peripheral blood from patients with active symptoms were all significantly higher compared with values obtained when patients were well. Compared with healthy control subjects, EDN concentrations in the serum, plasma and urine from asymptomatic asthmatic patients, as well as EDN concentrations in the serum and plasma from patients with mild atopic dermatitis and asymptomatic patients with seasonal ***allergic*** ***rhinitis*** , were significantly higher. Blood and urine concentrations of EDN in patients with active symptoms, but not urine concentrations of EDN from symptomatic patients with seasonal ***allergic*** ***rhinitis*** , were significantly increased compared
with values obtained for healthy control subjects. The concentrations of EDN in the serum and plasma from bronchial asthmatic patients, serum from patients with atopic dermatitis and plasma from patients with mild atopic

dermatitis correlated with the number of eosinophils in peripheral blood; however, the concentration of EDN in the serum correlated with the number of eosinophils from asymptomatic patients with seasonal ***allergic*** ***rhinitis*** . Conclusions: Determination of blood and urinary concentrations of EDN is useful to monitor eosinophil activity in allergic diseases.

TI Concentrations of eosinophil-derived ***neurotoxin*** in the blood and urine of patients with allergic diseases.

AB Background: It has been reported that measurements of eosinophil-derived ***neurotoxin*** (EDN) may be useful to determine eosinophil activities in allergic diseases. Methods: Serum, plasma and urine concentrations of EDN in. . . well as EDN concentrations in the serum and plasma from patients with mild atopic dermatitis and asymptomatic patients with seasonal ***allergic*** ***rhinitis*** , were significantly higher. Blood and urine concentrations of EDN in patients with active symptoms, but not urine concentrations of EDN from symptomatic patients with seasonal ***allergic*** ***rhinitis*** , were significantly increased compared with values obtained for healthy control subjects. The concentrations of EDN in the serum and plasma. . . blood; however, the concentration of EDN in the serum correlated with the number of eosinophils from asymptomatic patients with seasonal ***allergic*** ***rhinitis*** . Conclusions: Determination of blood and urinary concentrations of EDN is useful to monitor eosinophil activity in allergic diseases.

IT . . .
immunology
Asthma (MeSH)

IT Diseases
atopic dermatitis: genetic disease, immune system disease, integumentary system disease, immunology
Dermatitis, Atopic (MeSH)

IT Diseases
seasonal ***allergic*** ***rhinitis*** : respiratory system disease, immunology
Rhinitis, Allergic, Perennial (MeSH)

IT Chemicals & Biochemicals
eosinophil-derived ***neurotoxin***

L5 ANSWER 20 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 7

AN 2004:897777 CAPLUS <<LOGINID::20100927>>

DN 142:5293

TI Lysozyme levels in the nasal secretions of patients with perennial ***allergic*** ***rhinitis*** and recurrent sinusitis

AU Kalfa, V. Cuneyt; Spector, Sheldon L.; Ganz, Tomas; Cole, Alexander M.

CS Division of Pediatric Immunology, Allergy, Rheumatology, Department of Pediatrics, University of California at Los Angeles, Los Angeles, CA, USA

SO Annals of Allergy, Asthma, & Immunology (2004), 93(3), 288-292
CODEN: ALAIF6; ISSN: 1081-1206

PB American College of Allergy, Asthma, & Immunology

DT Journal

LA English

AB The assocn. of perennial ***allergic*** ***rhinitis*** (PAR) with recurrent sinusitis (RS) is well recognized. Anatomical abnormalities at the osteomeatal complex or ciliary dysfunction may play a significant role in some patients. However, for most patients with allergy, the determinants of RS are unknown. To det. whether altered concns. of antimicrobial peptides and proteins, such as lysozyme, lactoferrin, human

.beta.-defensin-2 (HBD-2), and human neutrophil peptides 1 to 3 (HNP-1 to 3), contribute to the development of RS in patients with PAR. Nasal secretions were collected by vacuum aspiration from 15 individuals with PAR+RS, 16 with PAR alone, and 16 controls. Lysozyme and lactoferrin levels were detd. in nasal secretions by using quant. ELISA, and HBD-2 and HNP-1 to 3 levels were detd. in nasal secretions by using semiquant. Western blot anal. Eosinophil-derived ***neurotoxin*** (EDN) levels were measured by using ELISA as a marker of nasal eosinophilia in all 3 groups. Levels of EDN were elevated significantly in patients with PAR+RS compared with controls. Lysozyme levels were decreased significantly in patients with PAR+RS compared with PAR alone or controls. Mean lysozyme levels were significantly lower in patients with EDN levels greater than 1000 ng/mL vs. those with levels of 1000 ng/mL or less in the PAR+RS group. There were no statistically significant differences in lactoferrin, HBD-2, and HNP-1 to 3 levels among the 3 groups. The presence of eosinophils and their products and reduced lysozyme concns. may be crit. factors that predispose the airways of patients with PAR to RS.

OSC.G 6 THERE ARE 6 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

RE.CNT 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Lysozyme levels in the nasal secretions of patients with perennial ***allergic*** ***rhinitis*** and recurrent sinusitis

AB The assocn. of perennial ***allergic*** ***rhinitis*** (PAR) with recurrent sinusitis (RS) is well recognized. Anatomical abnormalities at the osteomeatal complex or ciliary dysfunction may play a. . . . ELISA, and HBD-2 and HNP-1 to 3 levels were detd. in nasal secretions by using semiquant. Western blot anal. Eosinophil-derived ***neurotoxin*** (EDN) levels were measured by using ELISA as a marker of nasal eosinophilia in all 3 groups. Levels of EDN. . . .

ST lysozyme nasal secretion ***allergic*** ***rhinitis*** sinusitis

IT Allergy
Inflammation
Nose, disease
(***allergic*** ***rhinitis*** ; lysozyme levels in nasal secretions of patients with perennial ***allergic*** ***rhinitis*** and recurrent sinusitis)

IT Eosinophilia
Human
(lysozyme levels in nasal secretions of patients with perennial ***allergic*** ***rhinitis*** and recurrent sinusitis)

IT Inflammation
Respiratory system, disease
(sinusitis; lysozyme levels in nasal secretions of patients with perennial ***allergic*** ***rhinitis*** and recurrent sinusitis)

IT 9001-63-2, Lysozyme
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(lysozyme levels in nasal secretions of patients with perennial ***allergic*** ***rhinitis*** and recurrent sinusitis)

L5 ANSWER 21 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 8

AN 2004:284019 CAPLUS <<LOGINID::20100927>>

DN 141:37280

TI Biochemical assessment of intracellular signal transduction pathways in eosinophils: implications for pharmacotherapy

AU Wong, Chun Kwok; Ip, Wai Ki; Lam, Christopher Wai Kei

CS Department of Chemical Pathology, Prince of Wales Hospital, The Chinese University of Hong Kong, Shatin, NT, Hong Kong
SO Critical Reviews in Clinical Laboratory Sciences (2004), 41(1), 79-113
CODEN: CRCLBH; ISSN: 1040-8363
PB CRC Press LLC
DT Journal; General Review
LA English
AB A review. Allergic asthma and ***allergic*** ***rhinitis*** are inflammatory diseases of the airway. Cytokines and chemokines produced by T helper (Th) type 2 cells (GM-CSF, IL-4, IL-5, IL-6, IL-9, IL-10 and IL-13), eotaxin, transforming growth factor-.beta., and IL-11 orchestrate most pathophysiol. processes of the late-phase allergic reaction, including the recruitment, activation, and delayed apoptosis of eosinophils, as well as eosinophilic degranulation to release eosinophilic cationic protein, major basic protein, and eosinophil-derived ***neurotoxin*** . These processes are regulated through an extensive network of interactive intracellular signal transduction pathways that have been intensively investigated recently. Our present review updates the cytokine and chemokine-mediated signal transduction mechanisms including the RAS-RAF-mitogen-activated protein kinases, Janus kinases (signal transducers and activators of transcription), phosphatidylinositol 3-kinase, nuclear factor-kappa B, activator protein-1, GATA, and cAMP-dependent pathways, and describes the roles of different signaling pathways in the regulation of eosinophil differentiation, recruitment, degranulation, and expression of adhesion mols. We shall also discuss different biochem. methods for the assessment of various intracellular signal transduction mols., and various antagonists of receptors, modulators, and inhibitors of intracellular signaling mols., many of which are potential therapeutic agents for treating allergic diseases.
OSC.G 12 THERE ARE 12 CAPLUS RECORDS THAT CITE THIS RECORD (12 CITINGS)
RE.CNT 192 THERE ARE 192 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB A review. Allergic asthma and ***allergic*** ***rhinitis*** are inflammatory diseases of the airway. Cytokines and chemokines produced by T helper (Th) type 2 cells (GM-CSF, IL-4, IL-5,. . . and delayed apoptosis of eosinophils, as well as eosinophilic degranulation to release eosinophilic cationic protein, major basic protein, and eosinophil-derived ***neurotoxin*** . These processes are regulated through an extensive network of interactive intracellular signal transduction pathways that have been intensively investigated recently.. . .
IT Allergy
Inflammation
Nose, disease
(***allergic*** ***rhinitis*** ; intracellular signal transduction pathways in eosinophils in allergies)

L5 ANSWER 22 OF 48 MEDLINE on STN
AN 2003235255 MEDLINE <<LOGINID::20100927>>
DN PubMed ID: 12757446
TI Nasal blockage and urinary leukotriene E4 concentration in patients with seasonal ***allergic*** ***rhinitis*** .
AU Higashi N; Taniguchi M; Mita H; Ishii T; Akiyama K
CS Clinical Research Center, National Sagamihara Hospital, Kanagawa, Japan.
SO Allergy, (2003 Jun) Vol. 58, No. 6, pp. 476-80.
Journal code: 7804028. ISSN: 0105-4538. L-ISSN: 0105-4538.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)

LA English
FS Priority Journals
EM 200310
ED Entered STN: 22 May 2003
Last Updated on STN: 10 Oct 2003
Entered Medline: 9 Oct 2003
AB BACKGROUND: Cysteinyl-leukotrienes have been reported to have a primary role in the induction of nasal blockage of ***allergic*** ***rhinitis*** . However, there has been little experimental evidence that substantiates the relationship between nasal blockage severity and urinary leukotriene E4 (U-LTE4) concentration in patients with seasonal ***allergic*** ***rhinitis*** (SAR). METHODS: The concentrations of urinary mediators in 20 SAR patients were measured using an enzyme immunoassay to determine the relationship between nasal blockage severity and U-LTE4 concentration in patients with SAR. RESULTS: The basal U-LTE4 concentration was significantly higher in SAR patients with severe nasal blockage than in those with mild nasal blockage and in healthy control subjects. Although U-LTE4 concentrationwas significantly higher in patients with both asthma and SAR than in SAR patients with mild nasal blockage, no significant difference in the U-LTE4 concentration between patients with both asthma and SAR and SAR patients with severe nasal blockage was found. There was a significant correlation between U-LTE4 and urinary 9alpha11beta-prostoglandin F2 (9alpha11betaPGF2) concentrations (rs = 0.51, P = 0.02) in SAR patients. CONCLUSIONS: Although specific sites and cells of cysteinyl-leukotriene biosynthesis could not be determined in this study, severe nasal blockage is associated with the increased excretion level of U-LTE4.
TI Nasal blockage and urinary leukotriene E4 concentration in patients with seasonal ***allergic*** ***rhinitis*** .
AB BACKGROUND: Cysteinyl-leukotrienes have been reported to have a primary role in the induction of nasal blockage of ***allergic*** ***rhinitis*** . However, there has been little experimental evidence that substantiates the relationship between nasal blockage severity and urinary leukotriene E4 (U-LTE4) concentration in patients with seasonal ***allergic*** ***rhinitis*** (SAR). METHODS: The concentrations of urinary mediators in 20 SAR patients were measured using an enzyme immunoassay to determine the. . .
CT . . . Check Tags: Female; Male
Adolescent
Adult
*Airway Obstruction: UR, urine
Case-Control Studies
Dinoprost: AA, analogs & derivatives
Dinoprost: UR, urine
*** Eosinophil-Derived Neurotoxin***
Humans
Immunoenzyme Techniques
*Leukotriene E4: UR, urine
Middle Aged
*Nasal Cavity
Osmolar Concentration
*Rhinitis, Allergic, Seasonal: PP, physiopathology
. . .
CN EC 3.1.- (Eosinophil-Derived ***Neurotoxin***); EC 3.1.- (Ribonucleases)

L5 ANSWER 23 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 9
AN 2003:204668 CAPLUS <<LOGINID::20100927>>
DN 139:5439
TI Increased Serum Concentration of Eosinophil-Derived ***Neurotoxin*** in Patients with Graves' Disease
AU Hidaka, Yoh; Kimura, Masahiro; Izumi, Yukiko; Takano, Toru; Tatsumi, Ke-Ita; Amino, Nobuyuki
CS Department of Laboratory Medicine, Osaka University Graduate School of Medicine, Osaka, Japan
SO Thyroid (2003), 13(2), 129-132
CODEN: THYRER; ISSN: 1050-7256
PB Mary Ann Liebert, Inc.
DT Journal
LA English
AB Eosinophil-derived ***neurotoxin*** (EDN) is released after activation and stimulation of eosinophils in allergic disease, which is a TH2-predominant condition. We previously reported that Graves' thyrotoxicosis develops or relapses after an attack of ***allergic*** ***rhinitis*** . In this study, to confirm the relation between Graves' disease and the allergic condition, we detd. the serum level of EDN in 30 untreated patients with Graves' disease, 50 patients with Hashimoto's thyroiditis, and 39 normal controls. Compared to the serum level in normal subjects (30.1 .+-. 15.6 ng/mL), EDN was increased in untreated patients with Graves' disease (52.4 .+-. 27.6 ng/mL), but not in patients with Hashimoto's thyroiditis (thyrotoxic, 30.9 .+-. 13.4 ng/mL; euthyroid, 30.0 .+-. 11.9 ng/mL; hypothyroid, 23.4 .+-. 10.2 ng/mL). A significant correlation was obsd. between the EDN level and the serum activity of TSH receptor antibody (r = 0.541, p < 0.0001). These data suggest that the allergic condition is closely related to Graves' disease and that a TH2-type immune response is crucial in the pathogenesis of Graves' disease.
OSC.G 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)
RE.CNT 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Increased Serum Concentration of Eosinophil-Derived ***Neurotoxin*** in Patients with Graves' Disease
AB Eosinophil-derived ***neurotoxin*** (EDN) is released after activation and stimulation of eosinophils in allergic disease, which is a TH2-predominant condition. We previously reported that Graves' thyrotoxicosis develops or relapses after an attack of ***allergic*** ***rhinitis*** . In this study, to confirm the relation between Graves' disease and the allergic condition, we detd. the serum level of. . .
ST Graves' disease allergy eosinophil derived ***neurotoxin***
IT Cytokines
RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
(EDN (eosinophil-derived ***neurotoxin***); increased serum concn. of eosinophil-derived ***neurotoxin*** in patients with Graves' disease)
IT Allergy
Inflammation
Nose, disease
(***allergic*** ***rhinitis*** ; increased serum concn. of eosinophil-derived ***neurotoxin*** in patients with Graves' disease in relation to ***allergic*** ***rhinitis***)
IT Antibodies and Immunoglobulins

RL: ADV (Adverse effect, including toxicity); BSU (Biological study, unclassified); BIOL (Biological study)
(autoantibodies; increased serum concn. of eosinophil-derived ***neurotoxin*** in patients with Graves' disease in relation to autoantibodies to TSH receptors)
IT T cell (lymphocyte)
(helper cell/inducer, TH2; increased serum concn. of eosinophil-derived ***neurotoxin*** in patients with Graves' disease)
IT Blood serum
Eosinophil
Graves' disease
Human
(increased serum concn. of eosinophil-derived ***neurotoxin*** in patients with Graves' disease)
IT Thyrotropin receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(increased serum concn. of eosinophil-derived ***neurotoxin*** in patients with Graves' disease)
IT Thyroid gland, disease
(thyrotoxicosis; increased serum concn. of eosinophil-derived ***neurotoxin*** in patients with Graves' disease)

L5 ANSWER 24 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 2002:301617 BIOSIS <<LOGINID::20100927>>
DN PREV200200301617
TI Increased total cellular eosinophil derived ***neurotoxin*** (EDN) in eosinophils (EOS) from patients with allergic asthma (AA).
AU Sedgwick, Julie B. [Reprint author]; Jansen, Kristyn J. [Reprint author]; Kita, Hirohito; Bartemes, Kathleen R.; Busse, William W. [Reprint author]
CS University of Wisconsin, Madison, WI, USA
SO Journal of Allergy and Clinical Immunology, (January, 2002) Vol. 109, No. 1 Supplement, pp. S227. print.
Meeting Info.: 58th Annual Meeting of the American Academy of Allergy, Asthma and Immunology. New York, NY, USA. March 01-06, 2002. American Academy of Allergy, Asthma, and Immunology.
CODEN: JACIBY. ISSN: 0091-6749.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 22 May 2002
Last Updated on STN: 22 May 2002
TI Increased total cellular eosinophil derived ***neurotoxin*** (EDN) in eosinophils (EOS) from patients with allergic asthma (AA).
IT . . .
Organisms
eosinophils: blood and lymphatics, immune system
IT Diseases
allergic asthma: immune system disease, respiratory system disease
Asthma (MeSH)
IT Diseases
allergic ***rhinitis*** : immune system disease, respiratory system disease
Rhinitis, Allergic, Perennial (MeSH)
IT Chemicals & Biochemicals
corticosteroids; total cellular eosinophil derived ***neurotoxin***

L5 ANSWER 25 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2002:493162 CAPLUS <<LOGINID::20100927>>
DN 137:139273
TI DNA microarray analysis of transforming growth factor-.beta. and related transcripts in nasal biopsies from patients with ***allergic*** ***rhinitis***
AU Benson, Mikael; Carlsson, Bjoern; Carlsson, Lena M. S.; Mostad, Petter; Svensson, Per-Arne; Cardell, Lars-Olaf
CS Allergy Laboratory, Department of Oto-Rhino-Laryngology, Malmoe University Hospital, Malmoe, Swed.
SO Cytokine+ (2002), 18(1), 20-25
CODEN: CYTIE9; ISSN: 1043-4666
PB Elsevier Science Ltd.
DT Journal
LA English
AB Decreased activity of anti-inflammatory cytokines like transforming growth factor (TGF)-.beta. may contribute to allergic inflammation. In vivo effects of TGF-.beta.-effects are difficult to infer from local concns., since TGF-.beta.-effects depend on a complex system of regulatory proteins and receptors. Instead the effects of TGF-.beta. might be inferred by examg. TGF-.beta.-inducible transcripts. In this study DNA microarrays were used to examine local expression of TGF-.beta., TGF-.beta.-regulatory and -inducible transcripts in nasal biopsies from patients with symptomatic ***allergic*** ***rhinitis*** and healthy controls. In addn., nasal fluids were analyzed with cytol. and immunol. methods. Nasal fluid eosinophils, albumin, eosinophil granulae proteins and IgE, but not TGF-.beta., were higher in patients than in controls. DNA microarray anal. of nasal mucosa showed expression of transcripts encoding TGF-.beta., TGF-.beta.-regulatory proteins and -receptors at variable levels in patients and controls. By comparison, anal. of 28 TGF-.beta.-inducible transcripts indicated that 23 of these had lower measurement values in patients than in controls, while one was higher, and the remaining four were absent in both patients and controls. In summary, TGF-.beta. and a complex system of regulatory genes and receptors are expressed in the nasal mucosa. Low expression of TGF-.beta.-inducible transcripts may indicate decreased TGF-.beta. activity in ***allergic*** ***rhinitis*** . DNA microarray anal. may be a way to study cytokine effects in vivo.
OSC.G 18 THERE ARE 18 CAPLUS RECORDS THAT CITE THIS RECORD (18 CITINGS)
RE.CNT 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI DNA microarray analysis of transforming growth factor-.beta. and related transcripts in nasal biopsies from patients with ***allergic*** ***rhinitis***
AB . . . microarrays were used to examine local expression of TGF-.beta., TGF-.beta.-regulatory and -inducible transcripts in nasal biopsies from patients with symptomatic ***allergic*** ***rhinitis*** and healthy controls. In addn., nasal fluids were analyzed with cytol. and immunol. methods. Nasal fluid eosinophils, albumin, eosinophil granulae. . . genes and receptors are expressed in the nasal mucosa. Low expression of TGF-.beta.-inducible transcripts may indicate decreased TGF-.beta. activity in ***allergic*** ***rhinitis*** . DNA microarray anal. may be a way to study cytokine effects in vivo.
ST DNA microarray transforming growth factor ***allergic*** ***rhinitis***
IT Eosinophil
Gene expression profiles, animal

Human
(DNA microarray anal. of transforming growth factor-.beta. and related transcripts in nasal biopsies from patients with ***allergic*** ***rhinitis***)

IT Albumins, biological studies
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(DNA microarray anal. of transforming growth factor-.beta. and related transcripts in nasal biopsies from patients with ***allergic*** ***rhinitis***)

IT Proteins
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(ECP (eosinophil cationic protein); DNA microarray anal. of transforming growth factor-.beta. and related transcripts in nasal biopsies from patients with ***allergic*** ***rhinitis***)

IT Cytokines
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(EDN (eosinophil-derived ***neurotoxin***); DNA microarray anal. of transforming growth factor-.beta. and related transcripts in nasal biopsies from patients with ***allergic*** ***rhinitis***)

IT Antibodies and Immunoglobulins
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(IgE; DNA microarray anal. of transforming growth factor-.beta. and related transcripts in nasal biopsies from patients with ***allergic*** ***rhinitis***)

IT Allergy
Inflammation
Nose, disease
(***allergic*** ***rhinitis*** ; DNA microarray anal. of transforming growth factor-.beta. and related transcripts in nasal biopsies from patients with ***allergic*** ***rhinitis***)

IT Nose
(mucosa; DNA microarray anal. of transforming growth factor-.beta. and related transcripts in nasal biopsies from patients with ***allergic*** ***rhinitis***)

IT Transforming growth factors
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(.beta.-; DNA microarray anal. of transforming growth factor-.beta. and related transcripts in nasal biopsies from patients with ***allergic*** ***rhinitis***)

L5 ANSWER 26 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN
AN 2001:935886 CAPLUS <<LOGINID::20100927>>
DN 136:66584
TI Rapid diagnostic method for distinguishing allergies and infections and nasal secretion collection unit
IN Kudla, Ronald; Small, Parker; Huang, Shih-Wen
PA University of Florida, USA
SO PCT Int. Appl., 43 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 4

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2001098783	A2	20011227	WO 2001-US16216	20010518
	WO 2001098783	A3	20020404		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,

CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 6551791	B1	20030422	US 2000-597360	20000619
AU 2001064700	A	20020102	AU 2001-64700	20010518
EP 1295128	A2	20030326	EP 2001-939150	20010518

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRAI US 2000-597360	A	20000619	
US 1995-576604	B2	19951221	
US 1996-621557	A2	19960325	
WO 1999-US5751	A2	19990316	
WO 2001-US16216	W	20010518	

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB A method and device for rapidly, non-invasively and inexpensively differentiating between ***allergic*** ***rhinitis*** , upper respiratory tract viral infection and bacterial sinusitis, comprises a support strip upon which is fixed discrete indicators of pH, protein content, nitrite content, leukocyte esterase activity, and eosinophil content or other measure of a substance found in allergic secretions, such as TAME esterase, of a sample with which said reagent test strip is contacted. Contact of a nasal secretion with the device of this invention permits differentiation between allergic, bacterial and viral conditions, based on pH, protein content, leukocyte esterase activity, nitrite content, eosinophil content and TAME esterase activity. The invention further provides a novel means for collecting nasal secretions to facilitate differential diagnosis of sinusitis, upper respiratory tract viral infection and ***allergic*** ***rhinitis*** .

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB A method and device for rapidly, non-invasively and inexpensively differentiating between ***allergic*** ***rhinitis*** , upper respiratory tract viral infection and bacterial sinusitis, comprises a support strip upon which is fixed discrete indicators of pH,. . . provides a novel means for collecting nasal secretions to facilitate differential diagnosis of sinusitis, upper respiratory tract viral infection and ***allergic*** ***rhinitis*** .

ST diagnosis allergy infection nasal secretion collection app; ***allergic*** ***rhinitis*** diagnosis nasal secretion; upper respiratory tract viral infection diagnosis; bacterial sinusitis diagnosis nasal secretion; esterase TAME ***allergic*** ***rhinitis*** infection diagnosis

IT Cytokines
RL: ANT (Analyte); DGN (Diagnostic use); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(EDN (eosinophil-derived ***neurotoxin***); rapid diagnostic method for distinguishing allergies and infections and nasal secretion collection unit)

IT Allergy
Inflammation
Nose, disease
(***allergic*** ***rhinitis*** ; rapid diagnostic method for

distinguishing allergies and infections and nasal secretion collection unit)

L5 ANSWER 27 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 10
AN 2002:409682 CAPLUS <<LOGINID::20100927>>
DN 137:230874
TI Late type of the nasal allergic response
AU Pelikan, Z.
CS Department of Allergology and Immunology, Institute of Medical Sciences "De Klokkenberg", Breda, 4836 AE, Neth.
SO Scripta Medica Facultatis Medicae Universitatis Brunensis Masarykianae (2001), 74(5), 303-343
CODEN: SMFME9; ISSN: 1211-3395
PB Masarykova Univerzita v Brne, Lekarska Fakulta
DT Journal; General Review
LA English
AB A review. Three types of allergy component, i.e., Type I (immediate), Type III (late) and Type IV (delayed), can be involved in ***allergic*** ***rhinitis*** . Patients with nasal allergy, when challenged with an allergen during the nasal provocation tests, may develop different types of nasal response: immediate (INR), late (LNR) or delayed (DYNR). LNR occurs in 41% of the rhinitis patients. The clin. course of LNR, as recorded by rhinomanometry, is as follows: onset within 4 to 8 h, max. within 6 to 12 h and resolving within 24 h of the challenge with an allergen. LNR occurs either in an isolated form or in combination with INR. LNR is accompanied by severe nasal obstruction, while hypersensitivity, sneezing and itching appear to a lesser degree. LNR is regularly assocd. with other diagnostic parameters, such as, pos. disease history (in 23%), rhinoscopic changes (in 90%, violaceous nasal musoca, in some cases also small mucosal haemorrhages), pos. late skin response (in 65%, mostly induration), increased serum concn. of the total IgG (in 51%), increased blood oesinophilia (in 23%) and increased blood leukocyte counts (in 13%). LNR can also be accompanied by secondary responses of other organs (headache, palpebral edema, conjunctivitis, otitis media, sinusitis, bronchial obstruction and general malaise symptoms). Pos. LNR is accompanied by changes in the counts of various cell types in nasal secretions, such as, neutrophils (84%), eosinophils (58%), epithelial cells (73%), goblet cells (63%), basophils (8%) and lymphocytes (6%). Nasal mucosa biopsy during pos. LNR reveals an edematous epithelium with damaged integrity, sporadic breaches filled with fluid, irregular compactness of the basement membrane, an edematous lamina propria contg. eosinophilneutrophil infiltrates, perivascular oeadema and dilatation of mucosal capillaries. LNR may also be accompanied by the appearance of total IgG in nasal secretions (46%) and an increase in concns. of some compds. in nasal secretions (kinins, TAME-esterase, LTB4, LTC4, LTD4, LTE4, MBP, ECP, NCF, PGF2.alpha., histamine and eosinophil-derived ***neurotoxin***) LNR has also been recorded after food ingestion challenge. Pos. LNR can significantly be prevented by topical (intranasal) application of disodium cromoglycate, glucocorticosteroids or nedocromil sodium, whereas H1- and H2-receptor antagonists and immunotherapy have no significant effects on LNR. A possible involvement of various components on the late hypersensitivity mechanism(s) (Type III) in clin. LNR cannot be excluded.
OSC.G 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
RE.CNT 131 THERE ARE 131 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB . . . Three types of allergy component, i.e., Type I (immediate), Type

III (late) and Type IV (delayed), can be involved in ***allergic*** ***rhinitis*** . Patients with nasal allergy, when challenged with an allergen during the nasal provocation tests, may develop different types of nasal. . . concns. of some compds. in nasal secretions (kinins, TAME-esterase, LTB4, LTC4, LTD4, LTE4, MBP, ECP, NCF, PGF2.alpha., histamine and eosinophil-derived ***neurotoxin***) LNR has also been recorded after food ingestion challenge. Pos. LNR can significantly be prevented by topical (intranasal) application of. . .

ST review ***allergic*** ***rhinitis*** antihistamine immunotherapy hemorrhage

IT Allergy
Inflammation
Nose, disease
(***allergic*** ***rhinitis*** ; late type of nasal allergic response)

L5 ANSWER 28 OF 48 EMBASE

AN 2001004496 EMBASE <<LOGINID::20100927>>

TI The relationship of sputum eosinophilia and sputum cell generation of IL-5.

AU Liu, Lin Ying; Swensen, Cheri A.; Kelly, Elizabeth A.Becky; Kita, Hirohito; Busse, William W., Dr. (correspondence)

CS Allergy and Immunology and bPulmonary and Critical Care Sections of the Department of Medicine, University of Wisconsin, Madison.

AU Busse, William W., Dr. (correspondence)

CS Section of Allergy and Immunology, Univ. of Wisconsin School of Med., CSC H6/367, 600 Highland Ave, Madison, WI 53972, United States.

SO Journal of Allergy and Clinical Immunology, (2000) Vol. 106, No. 6, pp. 1063-1069.
Refs: 30
ISSN: 0091-6749 CODEN: JACIBY

CY United States

DT Journal; Article

FS 026 Immunology, Serology and Transplantation

LA English

SL English

ED Entered STN: 11 Jan 2001
Last Updated on STN: 11 Jan 2001

AB Background: Eosinophil recruitment to the airway after antigen challenge is regulated by many factors, including airway cell generation of cytokines. Objectives: The purpose of this study was to determine the relationship between sputum cell generation of IL-5 and the appearance of eosinophils in the sputum after antigen challenge. Method: Sputum samples from 11 allergic subjects were collected before and again 4 and 24 hours after antigen challenge. In 6 of these subjects, induced sputum samples were also obtained 48 hours and 7 days after challenge. Sputum leukocyte differential and cell counts and eosinophil-derived ***neurotoxin*** levels were determined. Sputum cells were then cultured with PHA (10 .mu.g/mL) to stimulate IL-5 and IFN-.gamma., which were measured in culture supernatants. Result: An increase in sputum eosinophils and eosinophil-derived ***neurotoxin*** levels was detected at 4 hours after antigen challenge, with peak values at 24 hours. In contrast, significant increases in ex vivo generation of IL-5 by sputum cells was not seen until 24 hours after challenge. At 24 hours, PHA-induced IL-5 correlated with airspace eosinophil values (r(s) = 0.78, P < .01). In addition, the ratio of IFN-.gamma./IL-5 decreased at 24 hours (P < .05)

and had an inverse correlation with sputum eosinophils (r(s) = -0.68, P < .05). Conclusion: Although eosinophils are increased in the airway lumen as early as 4 hours, the ex vivo generation of IL-5 by sputum cells is first noted in samples obtained 24 hours after antigen challenge. This suggests that the early (4 hours) recruitment of eosinophils to the airway lumen may be regulated by factors other than IL-5 or that mucosal cells (rather than airspace cells) contribute to the IL-5 generation at this time point. Furthermore, IL-5 generation by airspace cells may be more responsible for either eosinophil recruitment or retention at later time points.

AB . . . sputum samples were also obtained 48 hours and 7 days after challenge. Sputum leukocyte differential and cell counts and eosinophil-derived ***neurotoxin*** levels were determined. Sputum cells were then cultured with PHA (10 .mu.g/mL) to stimulate IL-5 and IFN-.gamma., which were measured in culture supernatants. Result: An increase in sputum eosinophils and eosinophil-derived ***neurotoxin*** levels was detected at 4 hours after antigen challenge, with peak values at 24 hours. In contrast, significant increases in. . .

CT Medical Descriptors:
adult
****allergic rhinitis***
*allergy
antigen recognition
article
*asthma
clinical article
eosinophil
*eosinophilia
female
human
lung lavage
male
priority journal
sputum analysis
*interleukin 5

L5 ANSWER 29 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 11

AN 2000:470648 BIOSIS <<LOGINID::20100927>>

DN PREV200000470648

TI Mechanism of topical glucocorticoid treatment of hay fever: IL-5 and eosinophil activation during natural allergen exposure are suppressed, but IL-4, IL-6, and IgE antibody production are unaffected.

AU Kita, Hirohito [Reprint author]; Jorgensen, Ronald K.; Reed, Charles E.; Dunnette, Sandra L.; Swanson, Mark C.; Bartemes, Kathleen R.; Squillace, Diane; Blomgren, Judy; Bachman, Kay; Gleich, Gerald J.

CS Department of Immunology, Mayo Clinic and Foundation, Rochester, MN, 55905, USA

SO Journal of Allergy and Clinical Immunology, (September, 2000) Vol. 106, No. 3, pp. 521-529. print.
CODEN: JACIBY. ISSN: 0091-6749.

DT Article

LA English

ED Entered STN: 1 Nov 2000
Last Updated on STN: 10 Jan 2002

AB Background: ***Allergic*** ***rhinitis*** is traditionally defined as an IgE- and mast cell-mediated hypersensitivity reaction. Allergen

challenge models suggest that cytokines and eosinophil mediators may also play roles. However, the causal relationship among inflammatory cells, their products, and patients' symptoms during natural allergen exposure has not been established. Objective: We sought to elucidate the mechanisms of seasonal ***allergic*** ***rhinitis*** and the beneficial effects of topical glucocorticoids. Methods: Thirty patients with ragweed-induced hay fever and a strongly positive serologic test response for ragweed IgE antibody received budesonide nasal spray or placebo in a randomized, parallel, double-blind study. Nasal wash fluids and sera were collected before and during the hay fever season. The levels of inflammatory mediators and allergen-specific immunoglobulins were measured by immunoassay. The activation markers on blood eosinophils were quantitated by flow cytometry. Results: Compared with placebo-treated patients, budesonide-treated patients had strikingly reduced symptoms. In the placebo group, nasal symptoms correlated with nasal lavage fluid eosinophil-derived ***neurotoxin*** and IL-5 levels. At the season peak, the budesonide-treated group had significantly lower nasal fluid eosinophil-derived ***neurotoxin*** , IL-5, and soluble intracellular adhesion molecule-1 levels. In the treated group eosinophil expression of CD11b was suppressed at the season peak. In contrast, levels of IL-4 and IL-6 in nasal fluid and the seasonal increases in serum ragweed-specific IgE and nasal fluid IgA antibodies did not differ between groups. Conclusion: Eosinophilic inflammation plays a critical role in seasonal ***allergic*** ***rhinitis*** symptoms. One of the therapeutic effects of glucocorticoids is to suppress this inflammation.

AB Background: ***Allergic*** ***rhinitis*** is traditionally defined as an IgE- and mast cell-mediated hypersensitivity reaction. Allergen challenge models suggest that cytokines and eosinophil mediators. . . and patients' symptoms during natural allergen exposure has not been established. Objective: We sought to elucidate the mechanisms of seasonal ***allergic*** ***rhinitis*** and the beneficial effects of topical glucocorticoids. Methods: Thirty patients with ragweed-induced hay fever and a strongly positive serologic test. . . placebo-treated patients, budesonide-treated patients had strikingly reduced symptoms. In the placebo group, nasal symptoms correlated with nasal lavage fluid eosinophil-derived ***neurotoxin*** and IL-5 levels. At the season peak, the budesonide-treated group had significantly lower nasal fluid eosinophil-derived ***neurotoxin*** , IL-5, and soluble intracellular adhesion molecule-1 levels. In the treated group eosinophil expression of CD11b was suppressed at the season. . . IgE and nasal fluid IgA antibodies did not differ between groups. Conclusion: Eosinophilic inflammation plays a critical role in seasonal ***allergic*** ***rhinitis*** symptoms. One of the therapeutic effects of glucocorticoids is to suppress this inflammation.

IT . . .
Medicine, Medical Sciences); Pharmacology

IT Parts, Structures, & Systems of Organisms
eosinophil: blood and lymphatics, immune system, activation

IT Diseases
allergic ***rhinitis*** : immune system disease, respiratory system disease
Rhinitis, Allergic, Perennial (MeSH)

IT Diseases
hay fever: immune system disease, respiratory system disease, ragweed-induced
Hay. . .

CD-11b: expression; IL-4 [interleukin-4]: antibody production; IL-5 [interleukin-5]; IL-6 [interleukin-6]: antibody production; allergen: exposure; budesonide: antiallergic-drug, nasal spray; cytokine; eosinophil-derived ***neurotoxin*** ; glucocorticoids: topical; immunoglobulin E: antibody production; immunoglobulins: allergen-specific; inflammatory mediators; ragweed: allergen; soluble intracellular adhesion molecule-1

L5 ANSWER 30 OF 48 LIFESCI COPYRIGHT 2010 CSA on STN
AN 2001:78565 LIFESCI <<LOGINID::20100927>>
TI Mechanism of topical glucocorticoid treatment of hay fever: IL-5 and eosinophil activation during natural allergen exposure are suppressed, but IL-4, IL-6, and IgE antibody production are unaffected
AU Kita, H.; Jorgensen, R.K.; Reed, C.E.; Dunnette, S.L.; Swanson, M.C.; Bartemes, K.R.; Squillace, D.; Blomgren, J.; Bachman, K.; Gleich, G.J.
CS Department of Immunology, Mayo Clinic and Foundation, Rochester, MN 55905, USA
SO Journal of Allergy and Clinical Immunology [J. Allergy Clin. Immunol.], (20000900) vol. 106, no. 3, Pt. 1, pp. 521-9.
ISSN: 0091-6749.
DT Journal
FS F
LA English
SL English
AB ***Allergic*** ***rhinitis*** is traditionally defined as an IgE- and mast cell-mediated hypersensitivity reaction. Allergen challenge models suggest that cytokines and eosinophil mediators may also play roles. However, the causal relationship among inflammatory cells, their products, and patients' symptoms during natural allergen exposure has not been established. We sought to elucidate the mechanisms of seasonal ***allergic*** ***rhinitis*** and the beneficial effects of topical glucocorticoids. Thirty patients with ragweed-induced hay fever and a strongly positive serologic test response for ragweed IgE antibody received budesonide nasal spray or placebo in a randomized, parallel, double-blind study. Nasal wash fluids and sera were collected before and during the hay fever season. The levels of inflammatory mediators and allergen-specific immunoglobulins were measured by immunoassay. The activation markers on blood eosinophils were quantitated by flow cytometry. Compared with placebo-treated patients, budesonide-treated patients had strikingly reduced symptoms. In the placebo group, nasal symptoms correlated with nasal lavage fluid eosinophil-derived ***neurotoxin*** and IL-5 levels. At the season peak, the budesonide-treated group had significantly lower nasal fluid eosinophil-derived ***neurotoxin*** , IL-5, and soluble intracellular adhesion molecule-1 levels. In the treated group eosinophil expression of CD11b was suppressed at the season peak. In contrast, levels of IL-4 and IL-6 in nasal fluid and the seasonal increases in serum ragweed-specific IgE and nasal fluid IgA antibodies did not differ between groups. Eosinophilic inflammation plays a critical role in seasonal ***allergic*** ***rhinitis*** symptoms. One of the therapeutic effects of glucocorticoids is to suppress this inflammation.
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L5 ANSWER 31 OF 48 MEDLINE on STN
AN 1999308481 MEDLINE <<LOGINID::20100927>>
DN PubMed ID: 10380774
TI Basophil histamine release, IgE, eosinophil counts, ECP, and EPX are related to the severity of symptoms in seasonal ***allergic*** ***rhinitis*** .
AU Winther L; Moseholm L; Reimert C M; Stahl Skov P; Kaergaard Poulsen L
CS Allergy Unit, National University Hospital, Copenhagen, Denmark.
SO Allergy, (1999 May) Vol. 54, No. 5, pp. 436-45.
Journal code: 7804028. ISSN: 0105-4538. L-ISSN: 0105-4538.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 199909
ED Entered STN: 25 Sep 1999
Last Updated on STN: 25 Sep 1999
Entered Medline: 3 Sep 1999
AB BACKGROUND: Serum specific IgE, basophil histamine release, and blood eosinophil parameters are associated with ***allergic*** ***rhinitis*** , but investigations of the relationship to the severity of allergic symptoms are few and conflicting. Our study aimed to investigate the seasonal changes in the following laboratory tests: specific IgE, basophil histamine release, eosinophil counts, and serum and plasma eosinophil cationic protein (ECP) and eosinophil protein X (EPX), and to analyze, in detail, the relationship of each individual test to the severity of symptoms in rhinitis patients allergic to both birch and grass pollen. METHODS: The above tests were performed on blood samples obtained from 49 ***allergic*** ***rhinitis*** patients during the birch-pollen season, during the grass-pollen season, and after the seasons. Symptom-medication diaries were filled in during both pollen seasons. We used partial least square (PLS) analysis to establish an optimal statistical link between the symptom score and medication and the laboratory tests, in an investigator-independent way. RESULTS: Increases in specific IgE, basophil histamine release, eosinophil counts, serum ECP and EPX, and plasma EPX were observed from the birch-pollen season to the grass-pollen season, followed by a decrease from the grass-pollen season to after the pollen seasons, except for the specific IgE. No seasonal changes in plasma ECP and total IgE were seen. The PLS analysis found a relationship between symptom score and medication and the aggregate laboratory tests (F-test value 40.2, correlation 0.34 for the cumulative relation). However, the variation in laboratory tests could explain only

half of the total variation in symptoms and less than a quarter of the total variation in medication. The symptom score and, to a minor degree, medication were especially correlated with the basophil histamine-release results, with a decreasing relevance of specific IgE, eosinophil counts, total IgE, serum and plasma EPX, and serum ECP. Plasma ECP was not related to the symptom score and medication. CONCLUSIONS: A significant relationship between the severity of ***allergic*** ***rhinitis*** and various allergic inflammatory markers was found but could account for only a minor part of the variation in the patients' evaluation of their disease.

TI Basophil histamine release, IgE, eosinophil counts, ECP, and EPX are related to the severity of symptoms in seasonal ***allergic*** ***rhinitis*** .

AB BACKGROUND: Serum specific IgE, basophil histamine release, and blood eosinophil parameters are associated with ***allergic*** ***rhinitis*** , but investigations of the relationship to the severity of allergic symptoms are few and conflicting. Our study aimed to investigate. . . patients allergic to both birch and grass pollen. METHODS: The above tests were performed on blood samples obtained from 49 ***allergic*** ***rhinitis*** patients during the birch-pollen season, during the grass-pollen season, and after the seasons. Symptom-medication diaries were filled in during both. . . ECP. Plasma ECP was not related to the symptom score and medication. CONCLUSIONS: A significant relationship between the severity of ***allergic*** ***rhinitis*** and various allergic inflammatory markers was found but could account for only a minor part of the variation in the. . .

CT Check Tags: Female; Male
Adolescent
Adult
*Basophils: PH, physiology
Blood Proteins: AN, analysis
Eosinophil Granule Proteins
*** Eosinophil-Derived Neurotoxin***
*Eosinophils: PH, physiology
Histamine Release
Humans
Immunoglobulin E: BL, blood
*Inflammation Mediators: AN, analysis
Leukocyte Count
Middle Aged
. . .

CN 0 (Blood Proteins); 0 (Eosinophil Granule Proteins); 0 (Inflammation Mediators); EC 3.1.- (Eosinophil-Derived ***Neurotoxin***); EC 3.1.- (Ribonucleases)

L5 ANSWER 32 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 12

AN 1999:445084 BIOSIS <<LOGINID::20100927>>

DN PREV199900445084

TI Novel co-operation between eotaxin and substance-P in inducing eosinophil-derived ***neurotoxin*** release.

AU El-Shazly, Amr [Reprint author]; Ishikawa, Takeru

CS Department of Otolaryngology and Head and Neck Surgery, St. George's Hospital - London University, Blackshaw Road, London, UK

SO Mediators of Inflammation, (1999) Vol. 8, No. 3, pp. 177-179. print. ISSN: 0962-9351.

DT Article

LA English
ED Entered STN: 26 Oct 1999
Last Updated on STN: 26 Oct 1999
AB Eosinophils, chemokines, and neuropeptides are thought to play effector roles in the pathogenesis of allergic diseases such as rhinitis. Eotaxin is a novel C-C chemokine with a potent and relatively specific eosinophil chemoattractant activity that binds selectively to CCR3 receptor, however, its activity in inducing eosinophil granules proteins release is poorly characterized. This study was performed to determine whether eotaxin primes eosinophil exocytosis and whether this co-operates with the sensory neuroimmune-axis. In the present communication, we show that 10 ng/ml eotaxin primed normal human eosinophil for exaggerated eosonophil-derived ***neurotoxin*** (EDN) release stimulated by 10-8 M Substance-P (SP). This novel priming was blocked by; 7B11 and Herbimycin A (HA), the CCR3 antagonist and the tyrosine kinase inhibitor, respectively. SDS-Page studies showed significant tyrosine phosphorylation of several protein residues induced by 1-8 M SP only after priming with 10 ng/ml eotaxin. These results demonstrate a novel co-operation between eotaxin and SP in inducing eosinophil cytotoxicity, which at least in part involves tyrosine kinases pathway(s).
TI Novel co-operation between eotaxin and substance-P in inducing eosinophil-derived ***neurotoxin*** release.
AB. . . the sensory neuroimmune-axis. In the present communication, we show that 10 ng/ml eotaxin primed normal human eosinophil for exaggerated eosonophil-derived ***neurotoxin*** (EDN) release stimulated by 10-8 M Substance-P (SP). This novel priming was blocked by; 7B11 and Herbimycin A (HA), the. . .
IT . . .
(Chemical Coordination and Homeostasis)
IT Parts, Structures, & Systems of Organisms
eosinophil: blood and lymphatics, immune system, exocytosis
IT Diseases
allergic ***rhinitis*** : immune system disease, respiratory system disease
Rhinitis, Allergic, Perennial (MeSH)
IT Chemicals & Biochemicals
chemokines; eotaxin; ***neurotoxin*** : eosinophil-derived, release; substance P; C-C chemokine 3 receptor [CCR3]

L5 ANSWER 33 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 13
AN 1998:33451 BIOSIS <<LOGINID::20100927>>
DN PREV199800033451
TI Activated eosinophils elicit substance P release from cultured dorsal root ganglion neurons.
AU Garland, Allan; Necheles, Jonathan; White, Steven R.; Neeley, Scott P.; Leff, Alan R.; Carson, Shannon S.; Alger, Linda E.; McAllister, Kyron; Solway, Julian [Reprint author]
CS Univ. Chicago, MC 6026, 5841 S. Maryland Ave., Chicago, IL 60637, USA
SO American Journal of Physiology, (Nov., 1997) Vol. 273, No. 5 PART 1, pp. L1096-L1102. print.
CODEN: AJPHAP. ISSN: 0002-9513.
DT Article
LA English
ED Entered STN: 14 Jan 1998
Last Updated on STN: 14 Jan 1998
AB This study was performed to test the hypothesis that activated eosinophils

or their secretory products can directly stimulate sensory neurons to release their neuropeptides. Neurons derived from neonatal rat dorsal root ganglia (DRG), which synthesize and store sensory neuropeptides, were placed in primary cell culture and were exposed to eosinophils or their bioactive mediators. The resultant release of substance P (SP) was measured by enzyme-linked immunosorbent assay and was expressed as a percent (mean +- SE) of total neuronal SP content. Eosinophils were isolated from human volunteers with a history of ***allergic*** ***rhinitis*** and/or mild asthma and were activated by incubation with cytochalasin B (5 Mg/ml) and N-formylmethionyl-leucyl-phenylalanine (FMLP, 1 muM). Activated eosinophils (6 X 106/ml, suspended in Hanks' buffered salt solution (HBSS)) applied to cultured DRG neurons for 30 min increased basal SP release 2.4-fold compared with HBSS-exposed neurons (activated eosinophils 11.10 +- 2.48% vs. HBSS 4.59 +- 0.99%; P = 0.002), whereas neither nonactivated eosinophils nor cytochalasin B and FMLP in HBSS influenced SP release. Additional cultured DRG neurons were exposed to soluble products made by eosinophils. Compared with SP release under control conditions (2.37 +- 0.34%), major basic protein (MBP) increased release in a concentration-related fashion (e.g., 3 muM MBP: 6.23 +- 0.67%, P = 0.006 vs. control), whereas neither eosinophil cationic protein (3 muM), eosinophil-derived ***neurotoxin*** (3 muM), leukotriene D4 (500 nM), platelet-activating factor (100 nM), nor H2O2 (100 muM) affected SP release. These studies demonstrate that activated eosinophils can stimulate cultured DRG neurons directly and suggest that MBP may be the responsible mediator.

AB. . . a percent (mean +- SE) of total neuronal SP content. Eosinophils were isolated from human volunteers with a history of ***allergic*** ***rhinitis*** and/or mild asthma and were activated by incubation with cytochalasin B (5 Mg/ml) and N-formylmethionyl-leucyl-phenylalanine (FMLP, 1 muM). Activated eosinophils. . . (e.g., 3 muM MBP: 6.23 +- 0.67%, P = 0.006 vs. control), whereas neither eosinophil cationic protein (3 muM), eosinophil-derived ***neurotoxin*** (3 muM), leukotriene D4 (500 nM), platelet-activating factor (100 nM), nor H2O2 (100 muM) affected SP release. These studies demonstrate. . .

L5 ANSWER 34 OF 48 SCISEARCH COPYRIGHT (c) 2010 The Thomson Corporation on STN

AN 1997:842733 SCISEARCH <<LOGINID::20100927>>

GA The Genuine Article (R) Number: YF568

TI Activated eosinophils elicit substance P release from cultured dorsal root ganglion neurons

AU Garland A (Reprint); Necheles J; White S R; Neeley S P; Leff A R; Carson S S; Alger L E; McAllister K; Solway J

CS UNIV CHICAGO, DEPT MED, PULM & CRIT CARE MED SECT, CHICAGO, IL 60637 USA; UNIV MED & DENT NEW JERSEY, ROBERT WOOD JOHNSON MED SCH, DEPT MED, DIV PULM & CRIT CARE MED, NEW BRUNSWICK, NJ 08903 USA

CYA USA

SO AMERICAN JOURNAL OF PHYSIOLOGY-LUNG CELLULAR AND MOLECULAR PHYSIOLOGY, (NOV 1997) Vol. 17, No. 5, pp. L1096-L1102.
ISSN: 1040-0605.

PB AMER PHYSIOLOGICAL SOC, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814.

DT Article; Journal

FS LIFE

LA English

REC Reference Count: 60

ED Entered STN: 1997
Last Updated on STN: 1997

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB This study was performed to test the hypothesis that activated eosinophils or their secretory products can directly stimulate sensory neurons to release their neuropeptides. Neurons derived from neonatal rat dorsal root ganglia (DRG), which synthesize and store sensory neuropeptides, were placed in primary cell culture and were exposed to eosinophils or their bioactive mediators. The resultant release of substance P (SP was measured by enzyme-linked immunosorbent assay and was expressed as a percent (mean +/- SE) of total neuronal SP content. Eosinophils were isolated from human volunteers with a history of ***allergic*** ***rhinitis*** and/or mild asthma and were activated by incubation with cytochalasin B (5 mu g/ml) and N-formylmethionyl-leucyl-phenylalanine (FMLP, 1 mu M). Activated eosinophils [6 x 10(6)/ml, suspended in Hanks' buffered salt solution (HBSS)] applied to cultured DRG neurons for 30 min increased basal SP release 2.4-fold compared with HBSS-exposed neurons (activated eosinophils 11.10 +/- 2.48% vs. HBSS 4.59 +/- 0.99%; P = 0.002), whereas neither nonactivated eosinophils nor cytochalasin B and FMLP in HBSS influenced SP release. Additional cultured DRG neurons were exposed to soluble products made by eosinophils. Compared with SP release under control conditions (2.37 +/- 0.34%), major basic protein (MBP) increased release in a concentration-related fashion (e.g., 3 mu M MBP: 6.23 +/- 0.67%, P = 0.006 vs. control), whereas neither eosinophil cationic protein (3 mu M), eosinophil-derived ***neurotoxin*** (3 mu M), leukotriene Dq (500 nM), platelet-activating factor (100 nM), nor H2O2 (100 mu M) affected SP release. These studies demonstrate that activated eosinophils can stimulate cultured DRG neurons directly and suggest that MBP may be the responsible mediator.

AB . . . a percent (mean +/- SE) of total neuronal SP content. Eosinophils were isolated from human volunteers with a history of ***allergic*** ***rhinitis*** and/or mild asthma and were activated by incubation with cytochalasin B (5 mu g/ml) and N-formylmethionyl-leucyl-phenylalanine (FMLP, 1 mu M).. . . mu M MBP: 6.23 +/- 0.67%, P = 0.006 vs. control), whereas neither eosinophil cationic protein (3 mu M), eosinophil-derived ***neurotoxin*** (3 mu M), leukotriene Dq (500 nM), platelet-activating factor (100 nM), nor H2O2 (100 mu M) affected SP release. These. . .

L5 ANSWER 35 OF 48 MEDLINE on STN

AN 1998100868 MEDLINE <<LOGINID::20100927>>

DN PubMed ID: 9438056

TI Clinical and nasal irrigation fluid findings in perennial ***allergic*** ***rhinitis*** .

AU Sulakvelidze I; Conway M; Evans S; Stetsko P I; Djuric V; Dolovich J

CS Department of Pediatrics, McMaster University, Hamilton, Ontario, Canada.

SO American journal of rhinology, (1997 Nov-Dec) Vol. 11, No. 6, pp. 435-41. Journal code: 8807268. ISSN: 1050-6586. L-ISSN: 1050-6586.

CY United States

DT (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

LA English

FS Priority Journals

EM 199802

ED Entered STN: 6 Mar 1998
Last Updated on STN: 3 Mar 2000
Entered Medline: 26 Feb 1998

AB Ten patients with perennial ***allergic*** ***rhinitis*** and 10 healthy subjects were studied to determine most discriminative nasal irrigation fluid marker(s) and to compare samples that were collected at baseline and over a 1-hour period, every 15 minutes. The latter were pooled and designated 1-hour sample. In the nasal irrigation we investigated the following inflammatory cells and soluble mediators: eosinophils, neutrophils, granulocyte-macrophage colony-stimulating factor, interleukin-4, interleukin-6, interleukin-8, ECP, EPX, MPO, leukotriene C4, leukotriene B4, prostaglandin E2, tryptase and fibrinogen. Patients with PAR were then treated for 2 weeks with the topical nasal steroid. The only marker that discriminated patients with perennial ***allergic*** ***rhinitis*** and healthy subjects was eosinophil count (EO%): correspondingly 14.01 +/- 5.8 and 0.18 +/- 0.09, (M +/- SD). Difference between the studied groups did not depend on the time of irrigation, baseline or 1-hour. EO% was also the only marker of a clinically successful treatment with the nasal steroid, 14.01 +/- 5.8 and 0.87 +/- 0.4, before and after treatment respectively. We conclude that EO% is the most sensitive inflammatory marker of perennial ***allergic*** ***rhinitis*** , and that baseline nasal irrigation can be used to study nasal mucosal inflammation.

TI Clinical and nasal irrigation fluid findings in perennial ***allergic*** ***rhinitis*** .

AB Ten patients with perennial ***allergic*** ***rhinitis*** and 10 healthy subjects were studied to determine most discriminative nasal irrigation fluid marker(s) and to compare samples that were. . . PAR were then treated for 2 weeks with the topical nasal steroid. The only marker that discriminated patients with perennial ***allergic*** ***rhinitis*** and healthy subjects was eosinophil count (EO%): correspondingly 14.01 +/- 5.8 and 0.18 +/- 0.09, (M +/- SD). Difference between. . . 0.87 +/- 0.4, before and after treatment respectively. We conclude that EO% is the most sensitive inflammatory marker of perennial ***allergic*** ***rhinitis*** , and that baseline nasal irrigation can be used to study nasal mucosal inflammation.

CT . . .

AN, analysis
Budesonide: AD, administration & dosage
Budesonide: TU, therapeutic use
Case-Control Studies
Chymases
Dinoprostone: AN, analysis
Eosinophil Granule Proteins
*** Eosinophil-Derived Neurotoxin***
Eosinophils: PA, pathology
Fibrinogen: AN, analysis
Follow-Up Studies
Glucocorticoids
Granulocyte-Macrophage Colony-Stimulating Factor: AN, analysis
Humans
Inflammation Mediators: AN,. . .

CN. . . Proteins); 0 (Eosinophil Granule Proteins); 0 (Glucocorticoids); 0 (Inflammation Mediators); 0 (Interleukin-6); 0 (Interleukin-8); EC 1.11.1.7 (Peroxidase); EC 3.1.- (Eosinophil-Derived ***Neurotoxin***); EC 3.1.- (Ribonucleases); EC 3.4.21.- (Serine Endopeptidases); EC 3.4.21.- (chymase 2); EC 3.4.21.39 (Chymases); EC 3.4.21.59 (Tryptases)

L5 ANSWER 36 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 14

AN 1997:263991 BIOSIS <<LOGINID::20100927>>
DN PREV199799570594
TI The effect of fluticasone propionate aqueous nasal spray on eosinophils and cytokines in nasal secretions of patients with ragweed ***allergic*** ***rhinitis*** .
AU Alvarado-Valdes, Carlos A.; Blomgren, Judith; Weiler, Deborah; Gleich, Gerald J.; Reed, Charles E. [Reprint author]; Field, Elizabeth A.; Wisniewski, Michael E.; Pobiner, Bonnie F.
CS Allergic Dis. Res. Lab., Mayo Clin., 200 First Street SW, Rochester, MN 55905, USA
SO Clinical Therapeutics, (1997) Vol. 19, No. 2, pp. 273-281.
CODEN: CLTHDG. ISSN: 0149-2918.
DT Article
LA English
ED Entered STN: 24 Jun 1997
Last Updated on STN: 24 Jun 1997
AB Cytokines active on eosinophils are important in the pathogenesis of allergic diseases. A study was conducted to determine if nasal eosinophilia in ***allergic*** ***rhinitis*** is associated with an increase in eosinophil-active cytokines in nasal secretions and to compare the effects of fluticasone propionate aqueous nasal spray with astemizole and placebo on the levels of these cytokines. Forty-five patients with moderately severe ragweed ***allergic*** ***rhinitis*** were randomly assigned to receive 2 weeks of treatment with fluticasone propionate aqueous nasal spray 200 mu-g once daily, astemizole 10 mg once daily, or placebo. Nasal lavage was performed in July (preseason), August (peak season), September (after 2 weeks of treatment), and October (postseason). The number of eosinophils, the amount of eosinophil-derived ***neurotoxin*** (EDN), and the amount of eosinophil survival-enhancing activity were measured. Total mean nasal symptom scores, concentrations of nasal eosinophils and EDN, and eosinophil survival-enhancing cytokine activity in nasal secretions were significantly lower after 2 weeks of treatment with fluticasone propionate compared with astemizole or placebo. Survival-enhancing activity was detected in the nasal secretions of 25 patients. By blocking activity with monoclonal antibodies, specific cytokines were identified (granulocyte macrophage-colony stimulating factor, 3 samples; interleukin-3, 2 samples; interleukin-5, 5 samples). In conclusion, eosinophil-active cytokine concentrations parallel the nasal symptoms of patients with ragweed ***allergic*** ***rhinitis*** . Unlike astemizole, fluticasone propionate significantly lowers cytokine activity in nasal tissue, which may contribute to the therapeutic efficacy of the drug.
TI The effect of fluticasone propionate aqueous nasal spray on eosinophils and cytokines in nasal secretions of patients with ragweed ***allergic*** ***rhinitis*** .
AB. . . on eosinophils are important in the pathogenesis of allergic diseases. A study was conducted to determine if nasal eosinophilia in ***allergic*** ***rhinitis*** is associated with an increase in eosinophil-active cytokines in nasal secretions and to compare the effects of fluticasone propionate aqueous nasal spray with astemizole and placebo on the levels of these cytokines. Forty-five patients with moderately severe ragweed ***allergic*** ***rhinitis*** were randomly assigned to receive 2 weeks of treatment with fluticasone propionate aqueous nasal spray 200 mu-g once daily, astemizole. . . August (peak season), September (after 2 weeks of treatment), and October (postseason). The number of eosinophils, the amount of eosinophil-derived

neurotoxin (EDN), and the amount of eosinophil survival-enhancing activity were measured. Total mean nasal symptom scores, concentrations of nasal eosinophils and. . . samples; interleukin-3, 2 samples; interleukin-5, 5 samples). In conclusion, eosinophil-active cytokine concentrations parallel the nasal symptoms of patients with ragweed ***allergic*** ***rhinitis*** . Unlike astemizole, fluticasone propionate significantly lowers cytokine activity in nasal tissue, which may contribute to the therapeutic efficacy of the. . .

IT Miscellaneous Descriptors
ANTIALLERGIC-DRUG; AQUEOUS NASAL SPRAY; ASTEMIZOLE; BLOOD AND LYMPHATICS; CYTOKINES; EFFICACY; EOSINOPHILS; FLUTICASONE PROPIONATE; IMMUNE SYSTEM; PATIENT; PHARMACOLOGY; RAGWEED ***ALLERGIC*** ***RHINITIS*** ; RESPIRATORY SYSTEM DISEASE

L5 ANSWER 37 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN DUPLICATE 15

AN 1996362971 EMBASE <<LOGINID::20100927>>

TI [Cell activation markers in rhinitis and rhinosinusitis: Part I: Eosinophil cationic protein].
ZELLAKTIVIERUNGSMARKER BEI RHINITIS UND RHINOSINUSITIS. TEIL 1: EOSINOPHILES KATIONISCHES PROTEIN (ECP).

AU Klimek, L., Dr. (correspondence)
CS HNO Universitatsklinik Mainz, Langenbeckstrasse 1, 55101 Mainz, Germany.
AU Rasp, G.
CS Klin./Poliklin. Hals-, Nasen-/O., LMU Munchen.

SO Laryngo- Rhino- Otologie, (1996) Vol. 75, No. 11, pp. 665-670.
Refs: 58
ISSN: 0935-8943 CODEN: LROTEX
CY Germany
DT Journal; Article
FS 011 Otorhinolaryngology
LA German
SL English; German
ED Entered STN: 23 Dec 1996
Last Updated on STN: 23 Dec 1996

AB Background: The quantitative analysis of the migration and activation of inflammatory cells is standard in current diagnosis of inflammatory diseases of the nasal mucosa. Histological and cytological examinations are mostly used for this purpose. Development and validation of assays for specific marlter proteins of the different cell populations make similar analyses possible from nasal secretion samples. Myeloperoxidase (MPO) or Elastase are important markers for neutrophil granulocytes as is Tryptase for mast cells, Lysozyme for macrophages/monocytes and Eosinophil Cationic Protein (ECP), Eosinophil ***Neurotoxin*** /Eosinophil Protein X (EDN/EPX) or Major Basic Protein (MBP) for eosinophil granulocytes. Methods: We performed a prospective study on healthy volunteers and patients with different inflammatory nasal and paranasal sinus diseases and analysed such cell activation markers in nasal secretions. In the healthy volunteers, 183 nasal secretion samples were obtained. A total of 515 samples were obtained in patients with the diagnosis: chronic sinusitis (n = 49), ***allergic*** ***rhinitis*** to perennial allergens (n = 94), chronic sinusitis with additional ***allergic*** ***rhinitis*** to perennial allergens (n = 36), nasal polyps (n = 28), ***allergic*** ***rhinitis*** to seasonal allergens extraseasonally (n = 131), and ***allergic*** ***rhinitis*** to seasonal allergens during the pollen season (n = 177). Results: In part I of this paper we describe the results for the Eosinophil Cationic Protein (ECP). In all

patients with active inflammatory reactions significantly higher ECP levels than in the controls were found. Moreover, ECP levels differed between the diseases investigated. Conclusions: ECP nasal secretion level seem to be a valuable marker for the assessment of nasal eosinophilic inflammation and might become an adjunct to current diagnostic measures.

AB . . . important markers for neutrophil granulocytes as is Tryptase for mast cells, Lysozyme for macrophages/monocytes and Eosinophil Cationic Protein (ECP), Eosinophil ***Neurotoxin*** /Eosinophil Protein X (EDN/EPX) or Major Basic Protein (MBP) for eosinophil granulocytes. Methods: We performed a prospective study on healthy volunteers. . . samples were obtained. A total of 515 samples were obtained in patients with the diagnosis: chronic sinusitis (n = 49), ***allergic*** ***rhinitis*** to perennial allergens (n = 94), chronic sinusitis with additional ***allergic*** ***rhinitis*** to perennial allergens (n = 36), nasal polyps (n = 28), ***allergic*** ***rhinitis*** to seasonal allergens extraseasonally (n = 131), and ***allergic*** ***rhinitis*** to seasonal allergens during the pollen season (n = 177).

Results: In part I of this paper we describe the. . .

CT Medical Descriptors:

adult

****allergic rhinitis: DI, diagnosis***

article

clinical trial

controlled study

*eosinophil

female

human

major clinical study

male

*nose polyp: DI, diagnosis

*nose secretion

*paranasal sinusitis: DI, diagnosis

priority journal

*eosinophil cationic protein: EC, endogenous. . .

L5 ANSWER 38 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 16

AN 1996:479038 BIOSIS <<LOGINID::20100927>>

DN PREV199699194294

TI Eosinophilia in the respiratory secretions of children with chronic respiratory diseases.

AU Wiersbitzky, S. K. W. [Reprint author]; Ballke, E.-H.; Mueller, C.; Bruns, R.; Heydolph, F.

CS Soldtmannstrasse 15, D-17478 Greifswald, Germany

SO Allergologie, (1996) Vol. 19, No. 7, pp. 310-315.
CODEN: ALLRDI. ISSN: 0344-5062.

DT Article

LA German

ED Entered STN: 24 Oct 1996
Last Updated on STN: 24 Oct 1996

AB Eosinophilia in the respiratory secretions of children with chronic respiratory diseases. The eosinophilic granulocytes are characteristic inflammatory cells in the respiratory mucosa of children and teenagers suffering from ***allergic*** ***rhinitis*** or allergic bronchial asthma. That is the basis for the concept of eosinophilic mucositis or eosinophilic bronchitis for such diseases in contrast to the neutrophilic

mucositis or neutrophilic (purulent) bronchitis due to viral or bacterial infections. By means of their aggressive metabolites (major basic protein (MBP), eosinophilic cationic protein (ECP), eosinophilic protein X (EPX), or eosinophil-derived ***neurotoxin*** (EDN), eosinophilic peroxidase (EPO)) the eosinophils play a central role in the pathophysiology of the transition from frequently relapsing obstructive bronchitis in early childhood due to infections to relapsing obstructive bronchitis of later childhood (= bronchial asthma) due to allergy, in most cases resulting from bronchial hyperreactivity. A significant secretory eosinophilia (i.e. more than 13% eosinophils in the cytological smears of nose, pharynx or the tracheo-bronchial wall), is an indicator for the existence of bronchial hyperreactivity, as a rule due to respiratory allergy. The intensity of the airway obstruction (nose, bronchus) does not correlate with the percentage of eosinophilia. Bronchoalveolar lavage (BAL) is not a suitable method for detecting secretary eosinophilia. Moreover, persistent eosinophilia of the respiratory secretions are a sensitive indicator for the continuous existence of inflammatory processes in the mucosa. Usually such cases require not only allergen elimination but also additional (topical) steroid administration. Bronchial asthma is under control only if the asthmatic symptoms and the lung function test have been normalized and the eosinophilia in the respiratory secretion has disappeared. The traditional counting of the eosinophils and the quantitative measurement of ECP give comparable results, but in many patients they can vary considerably. The counting of eosinophils should be given preference for routine cases (lower cost) whereas for large scale research the ECP determination can be more effective.

AB. . . chronic respiratory diseases. The eosinophilic granulocytes are characteristic inflammatory cells in the respiratory mucosa of children and teenagers suffering from ***allergic*** ***rhinitis*** or allergic bronchial asthma. That is the basis for the concept of eosinophilic mucositis or eosinophilic bronchitis for such diseases. . . By means of their aggressive metabolites (major basic protein (MBP), eosinophilic cationic protein (ECP), eosinophilic protein X (EPX), or eosinophil-derived ***neurotoxin*** (EDN), eosinophilic peroxidase (EPO)) the eosinophils play a central role in the pathophysiology of the transition from frequently relapsing obstructive. . .

IT Miscellaneous Descriptors
ALLERGIC BRONCHIAL ASTHMA; ***ALLERGIC*** ***RHINITIS*** ; ALLERGY; BLOOD AND LYMPHATIC DISEASE; CHILD; EOSINOPHILIA; IMMUNE SYSTEM DISEASE; INFLAMMATION; PULMONARY MEDICINE; RESPIRATORY SECRETIONS; RESPIRATORY SYSTEM DISEASE

L5 ANSWER 39 OF 48 EMBASE COPYRIGHT (c) 2010 Elsevier B.V. All rights reserved on STN DUPLICATE 17

AN 0009010559 EMBASE <<LOGINID::20100927>>

CP MEDLINE.RTM. is the source for the citation and abstract of this record.

TI Serum levels of eosinophil cationic protein and eosinophil protein x in pollen atopic patients with stable asthma and its relation with bronchial hyperresponsiveness..

AU Chivato, T. (correspondence); Martinez, D.; Blasco, R.; Melgarejo, M.; Gomez de Terreros, F.J.; Laguna, R.

CS Hospital del Aire, Madrid, Spain..

SO Allergologia et immunopathologia, (1996 Nov-Dec) Vol. 24, No. 6, pp. 243-247.
ISSN: 0301-0546

CY Spain

DT Journal; Article

FS MEDLINE
LA English
ED Entered STN: Mar 2010
Last Updated on STN: Mar 2010
AB Eosinophils are important effector cells in allergic inflammation described in ***allergic*** ***rhinitis*** (AR) and allergic bronchial asthma (BA). During the pollen season serum levels of eosinophil cationic protein (ECP) and eosinophil X protein/eosinophil-derived ***neurotoxin*** (EPX/EDN) are increased in BA. The aim of the present study was to evaluate the serum levels of ECP and EPC in pollen atopic patients with AR and BA during the winter. 92 patients were studied. They were divided into three groups: I 29 patients with AR, II 51 patients with BA and III 12 healthy subjects. ***Allergic*** ***rhinitis*** and bronchial asthma were diagnosed by routine clinical tests: clinical history, skin tests, total IgE and specific IgE. In addition ECP and EPX were determined in serum. All patients were asymptomatic, stable and without medical treatment. Methacholine challenge test (MCT) was performed in all patients. MCT were positive in 4 patients of group I and 45 patients of group II. ECP levels (ug/l) were: 21 (I), 24 (II) and 7 (III). EPX levels (ug/l) were 35 (I), 45 (II) and 21 (III). Statistical differences ($p < 0.01$) were observed both in ECP and EPX levels in patients with MCT positive in relation to patients with MCT negative, and in allergic patients (I and II) in comparison with the healthy subjects (III) ($p < 0.01$). ECP and EPX serum levels are increased in patients with a positive MCT in the winter, out of the pollen season, when patients are asymptomatic, stable and without treatment. This fact suggests that eosinophils play an important role in the pathogenesis of bronchial asthma.
AB Eosinophils are important effector cells in allergic inflammation described in ***allergic*** ***rhinitis*** (AR) and allergic bronchial asthma (BA). During the pollen season serum levels of eosinophil cationic protein (ECP) and eosinophil X protein/eosinophil-derived ***neurotoxin*** (EPX/EDN) are increased in BA. The aim of the present study was to evaluate the serum levels of ECP and. . . were divided into three groups: I 29 patients with AR, II 51 patients with BA and III 12 healthy subjects. ***Allergic*** ***rhinitis*** and bronchial asthma were diagnosed by routine clinical tests: clinical history, skin tests, total IgE and specific IgE. In addition. . .

L5 ANSWER 40 OF 48 SCISEARCH COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1996:392863 SCISEARCH <<LOGINID::20100927>>
GA The Genuine Article (R) Number: UL867
TI Distribution of eosinophil granule proteins in nasal mucosa of atopic patients with nasal polyposis
AU Min Y G (Reprint)
CS SEOUL NATL UNIV, COLL MED, DEPT OTORHINOLARYNGOL, 28 YONGON DONG, CHONGNO GU, SEOUL 110744, SOUTH KOREA (Reprint)
AU Kim Y J; Yun Y S
CYA SOUTH KOREA
SO ORL-JOURNAL FOR OTO-RHINO-LARYNGOLOGY AND ITS RELATED SPECIALTIES, (MAR-APR 1996) Vol. 58, No. 2, pp. 82-86.
ISSN: 0301-1569.
PB KARGER, ALLSCHWILERSTRASSE 10, CH-4009 BASEL, SWITZERLAND.
DT Article; Journal

FS CLIN
LA English
REC Reference Count: 15
ED Entered STN: 1996
Last Updated on STN: 1996
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Nasal polyposis is characterized by heavy eosinophilic infiltration into nasal polyp tissue. Nasal polyps have a predilection for the anteroinferior aspect of the middle turbinate and middle meatal area. To elucidate the pathogenesis of nasal polyps, the authors investigated the distribution of eosinophil infiltrating into nasal polyp tissue, especially at its pedicle, in comparison with the apparently normal nasal mucosa of the middle and inferior turbinates of the same patients. Tissue samples were taken from 12 ***allergic*** ***rhinitis*** patients with nasal polyps during endoscopic sinus surgery. Three kinds of monoclonal antibodies, EG1, EG2 and BMK-13, were used for immunohistochemical staining. The average number per high-power field of EG1+ cells was 6.33 at the pedicle of polyps and 4.68 and 4.36 at the middle and inferior turbinates, respectively; for EG2+ cells, it was 6.16 at the polyp pedicle and 2.06 and 2.47 at the middle and inferior turbinates, and for BMK-13+ cells, 4.20 at the polyp pedicle and 5.17 and 4.81 at the middle and inferior turbinates. There were no significant differences in the distribution of EG1+ and BMK-13+ cells, but a relatively larger number of activated eosinophils (EC2+ cells) was noted at the pedicle of polyps compared with the other sites ($p < 0.025$). In addition, the EG2+ cell/EG1+ cell ratio was significantly higher at the pedicle of polyps compared with the other sites ($p < 0.01$). The results of this study suggest that the activation of eosinophils is significant in the pathogenesis of nasal polyps in atopic patients rather than the number of eosinophils.
AB . . . apparently normal nasal mucosa of the middle and inferior turbinates of the same patients. Tissue samples were taken from 12 ***allergic*** ***rhinitis*** patients with nasal polyps during endoscopic sinus surgery. Three kinds of monoclonal antibodies, EG1, EG2 and BMK-13, were used for. . .
STP KeyWords Plus (R): MAJOR BASIC-PROTEIN; CATIONIC PROTEIN; ***NEUROTOXIN*** ; CELLS

L5 ANSWER 41 OF 48 EMBASE
AN 1997198322 EMBASE <<LOGINID::20100927>>
TI [Eosinophil cationic protein in allergic disease (part one)]. Proteina cationica del eosinofilo en enfermedades alergicas (Primera parte).
AU Miralles Lopez, J.C.; Negro, J.M.; Hernandez, J.; Caravaca, F.; Sarrio, F.
CS Plaza Juan XXIII, no. 3, 30008 Murcia, Spain.
AU Mirales Lopez, J.C. (correspondence)
CS Plaza Juan XXIII, no. 3, 30008 Murcia, Spain.
SO Revista Espanola de Alergologia e Inmunologia Clinica, (1996) Vol. 11, No. 2, pp. 77-84.
Refs: 47
ISSN: 0214-1477 CODEN: REACEN
CY Spain
DT Journal; General Review; (Review)
FS 026 Immunology, Serology and Transplantation
LA Spanish; Castilian
SL English; Spanish; Castilian

ED Entered STN: 7 Aug 1997
Last Updated on STN: 7 Aug 1997

AB The eosinophillic granulocyte, or eosinophil, is a proinflammatory cell which contains within its cytoplasmic granules many highly cytotoxic proteins, such as eosinophil cationic protein (ECP), ***neurotoxin*** , peroxidase and major basic protein. Upon stimulation of the cell these proteins, together with leukotrienes, prostaglandins and platelet activating factor (PAF) are released to the environment. ECP is a basic protein found in the matrix of the eosinophil's specific granule. It has cytotoxic and helmintotoxic activity and is a member of the multigenic ribonuclease family. Its amino acid sequence shows 66% homology to ***neurotoxin*** and 31% homology to human pancreatic ribonuclease. Although it is mainly expressed in eosinophils, ECP may also be found in variable amounts in basophils and neutrophils. EG2 monoclonal antibodies only detect the secreted form of ECP and thus identify activated eosinophils, whereas the EG1 antibodies detect both the secreted and the storage forms. IL-5, PAF and the c3a and c5a fractions of complement induce ECP release; there is also ECP release when eosinophils are incubated with anti-IgA and anti-IgG, but not upon incubation with specific allergens or anti-IgE. A close relationship has been demonstrated between deposits of eosinophil granular proteins and areas of epithelial cell destruction in the bronchi in several diseases including bronchial asthma; ECP further causes a dose-dependent increase in mucus secretion in airway fragments. ECP inhibits immunoglobulin production by human lymphoblastoid cell lines and the phytohemagglutinin-induced proliferation of human peripheral blood lymphocytes. Higher levels of ECP have been observed in serum and nasal lavage fluids in ***allergic*** ***rhinitis*** patients than in controls, in correlation with the intensity of the symptoms. A correlation has also been observed between ECP levels in nasal lavage fluids and non-specific nasal reactivity. Allergen challenge induces increase of the ECP levels in nasal lavage fluid. High levels of ECP have also been reported in some ocular disease, such as vernal keratoconjunctivitis and allergic keratoconjunctivitis with keratopathy.

AB . . . is a proinflammatory cell which contains within its cytoplasmic granules many highly cytotoxic proteins, such as eosinophil cationic protein (ECP), ***neurotoxin*** , peroxidase and major basic protein. Upon stimulation of the cell these proteins, together with leukotrienes, prostaglandins and platelet activating factor. . . and helmintotoxic activity and is a member of the multigenic ribonuclease family. Its amino acid sequence shows 66% homology to ***neurotoxin*** and 31% homology to human pancreatic ribonuclease. Although it is mainly expressed in eosinophils, ECP may also be found in. . . proliferation of human peripheral blood lymphocytes. Higher levels of ECP have been observed in serum and nasal lavage fluids in ***allergic*** ***rhinitis*** patients than in controls, in correlation with the intensity of the symptoms. A correlation has also been observed between ECP. . .

CT Medical Descriptors:
allergic rhinitis
*allergy
asthma
cell proliferation
eosinophil
human
keratoconjunctivitis
review
*eosinophil cationic protein: EC, endogenous compound

immunoglobulin: EC, endogenous compound
leukotriene: EC, endogenous compound
prostaglandin: EC, endogenous compound
thrombocyte activating factor: EC,. . .

L5 ANSWER 42 OF 48 CAPLUS COPYRIGHT 2010 ACS on STN DUPLICATE 18
AN 1993:253169 CAPLUS <<LOGINID::20100927>>
DN 118:253169
OREF 118:43970h,43971a
TI IL-5 is the predominant eosinophil-active cytokine in the antigen-induced pulmonary late-phase reaction
AU Ohnishi, T.; Kita, H.; Weiler, D.; Sur, S.; Sedgwick, J. B.; Calhoun, W. J.; Busse, W. W.; Abrams, J. S.; Gleich, G. J.
CS Dep. Immunol., Mayo Clin. Mayo Found., Rochester, MN, USA
SO American Review of Respiratory Disease (1993), 147(4), 901-7
CODEN: ARDSBL; ISSN: 0003-0805
DT Journal
LA English
AB The mechanism of airway eosinophilia during antigen-induced inflammation was investigated by measurement of eosinophil-active cytokines utilizing an eosinophil survival assay. In the first study, patients with ***allergic*** ***rhinitis*** underwent segmental bronchoprovocation (SBP) with low, medium, and high doses of ragweed ext. instilled into different bronchial subsegments; bronchoalveolar lavage (BAL) fluids were collected from each segment 12 min and 48 h after challenge. Eosinophil granule proteins and eosinophil survival activity were elevated in the 48-h (late-phase) BAL fluids from these segments. Correlations were obsd. between the concns. of eosinophil granule proteins and eosinophil survival activity in BAL fluids. Eosinophil survival activity was completely neutralized by anti-IL-5 monoclonal antibody in 5 of the 7 48 h samples tested representing 3 of the 4 patients. In the 2 remaining samples, eosinophil survival activity was only partially neutralized by either anti-IL-5 antibody or anti-granulocyte-macrophage colony-stimulating factor (GM-CSF) but was completely neutralized by anti-IL-5 and anti-GM-CSF in combination. Subsequently, in the second study, patients with ***allergic*** ***rhinitis*** were challenged by SBP with ragweed ext. Eosinophil survival activity was elevated in the 48 h BAL fluids; this activity was partially neutralized by anti-IL-5 antibody (48%) and completely neutralized by the combination of anti-IL-5 and anti-GM-CSF antibodies. Apparently, the eosinophil survival activity in the late inflammatory lesions following SBP with allergen is mainly assocd. with IL-5, with small contributions from GM-CSF. Thus, IL-5 is the predominant eosinophil-active cytokine present in BAL fluids during allergen-induced late-phase inflammation and may play a key role in the pathophysiol. of allergen-induced, eosinophil-predominant airway inflammation.
OSC.G 89 THERE ARE 89 CAPLUS RECORDS THAT CITE THIS RECORD (89 CITINGS)
AB . . . antigen-induced inflammation was investigated by measurement of eosinophil-active cytokines utilizing an eosinophil survival assay. In the first study, patients with ***allergic*** ***rhinitis*** underwent segmental bronchoprovocation (SBP) with low, medium, and high doses of ragweed ext. instilled into different bronchial subsegments; bronchoalveolar lavage. . . colony-stimulating factor (GM-CSF) but was completely neutralized by anti-IL-5 and anti-GM-CSF in combination. Subsequently, in the second study, patients with ***allergic*** ***rhinitis*** were challenged by SBP with ragweed ext. Eosinophil

survival activity was elevated in the 48 h BAL fluids; this activity. . .

IT Ragweed
(airway late-phase response to, eosinophilia in, in humans with ***allergic*** ***rhinitis*** , interleukin 5 in)

IT Proteins, specific or class
RL: PROC (Process)
(ECP (eosinophil cationic protein), release of, by eosinophil in airway late-phase response in humans with ***allergic*** ***rhinitis*** , interleukin 5 in relation to)

IT Lymphokines and Cytokines
RL: PROC (Process)
(EDN (eosinophil-derived ***neurotoxin***), release of, by eosinophil in airway late-phase response in humans with ***allergic*** ***rhinitis*** , interleukin 5 in relation to)

IT Lymphokines and Cytokines
RL: PROC (Process)
(MBP (major basic protein), release of, by eosinophil in airway late-phase response in humans with ***allergic*** ***rhinitis*** , interleukin 5 in relation to)

IT Eosinophil
(disease, eosinophilia, interleukin 5 mediation of, in airway late-phase response in humans with ***allergic*** ***rhinitis***)

IT Lymphokines and Cytokines
RL: BIOL (Biological study)
(interleukin 5, in airway eosinophil recruitment and activation in late-phase response in ***allergic*** ***rhinitis*** , in humans)

IT 83869-56-1, GM-CSF
RL: BIOL (Biological study)
(interleukin 5 and, in airway late-phase response in humans with ***allergic*** ***rhinitis***)

IT 9003-99-0, Eosinophil peroxidase
RL: PROC (Process)
(release of, by eosinophil in airway late-phase response in humans with ***allergic*** ***rhinitis*** , interleukin 5 in relation to)

L5 ANSWER 43 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 19

AN 1992:253594 BIOSIS <<LOGINID::20100927>>

DN PREV199293129919; BA93:129919

TI THE EOSINOPHIL AND ITS SIGNIFICANCE IN ALLERGIC DISEASES.

AU BRUIJNZEEL P L B [Reprint author]; RIHS S; BETZ S

CS SCHWEIZERISCHES INST ALLERGIE ASTHMAFORSCHUNG, OBERE STRASSE 22, CH-7270 DAVO PLATZ

SO Schweizerische Medizinische Wochenschrift, (1992) Vol. 122, No. 6, pp. 173-180.
CODEN: SMWOAS. ISSN: 0036-7672.

DT Article

FS BA

LA GERMAN

ED Entered STN: 23 May 1992
Last Updated on STN: 23 May 1992

AB Eosinophils are found in the blood and tissues of patients with allergic diseases such as asthma, ***allergic*** ***rhinitis*** and also atopic dermatitis. The number of eosinophils in the diseased tissue

generally correlates with the expression of clinical symptoms. Originally, the eosinophil was regarded as having an exclusively protective role, for example in host defense against parasites. More recently, the eosinophil is recognized as being a pro-inflammatory cell that can mediate allergic disease. The eosinophil is active in inflammation through the release of granule proteins and the synthesis and release of inflammatory mediators. The important eosinophil granule proteins are major basic protein (MBP), eosinophil cationic protein (ECP), eosinophil derived ***neurotoxin*** (EDN) and eosinophil peroxidase (EPO). These proteins have toxic effects on the surrounding tissue. Of additional importance for the allergic inflammatory reactions are membrane-derived mediators such as leukotriene C4 (LTC4), platelet activating factor (PAF) and Charcot-Leyden crystals. These mediators are synthesized and released after eosinophil activation, and act toxic on surrounding cells. Eosinophils are active in asthma, and increased numbers and eosinophil-derived mediator concentrations have been documented in bronchial biopsies, BAL and sputum. In addition, eosinophil granule proteins and inflammatory mediators are found in nasal secretions in ***allergic*** ***rhinitis*** . In atopic dermatitis one finds activated eosinophils and depositions of eosinophil granule proteins in skin biopsies. Eosinophils are not only active in mediating allergic inflammation, but interact in cellular networkd with antigen presenting cells (APC), mast cells, and T lymphocytes. These other cells influence eosinophil maturation, mobilization, tissue localization and activation.

AB Eosinophils are found in the blood and tissues of patients with allergic diseases such as asthma, ***allergic*** ***rhinitis*** and also atopic dermatitis. The number of eosinophils in the diseased tissue generally correlates with the expression of clinical symptoms.. . . release of inflammatory mediators. The important eosinophil granule proteins are major basic protein (MBP), eosinophil cationic protein (ECP), eosinophil derived ***neurotoxin*** (EDN) and eosinophil peroxidase (EPO). These proteins have toxic effects on the surrounding tissue. Of additional importance for the allergic. . . in bronchial biopsies, BAL and sputum. In addition, eosinophil granule proteins and inflammatory mediators are found in nasal secretions in ***allergic*** ***rhinitis*** . In atopic dermatitis one finds activated eosinophils and depositions of eosinophil granule proteins in skin biopsies. Eosinophils are not only. . .

IT Miscellaneous Descriptors
REVIEW HUMAN ANTIGEN PRESENTING CELLS MAST CELLS T-LYMPHOCYTES MAJOR BASIC PROTEIN EOSINOPHIL CATIONIC PROTEIN EOSINOPHIL-DERIVED ***NEUROTOXIN*** EOSINOPHIL PEROXIDASE LEUKOTRIENE C-4 PLATELET ACTIVATING FACTOR CHARCOT-LEYDEN CRYSTALS DERMATITIS ATOPY INFLAMMATION ASTHMA RHINITIS

L5 ANSWER 44 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 20

AN 1992:91410 BIOSIS <<LOGINID::20100927>>

DN PREV199293047960; BA93:47960

TI IMMEDIATE AND LATE AIRWAY RESPONSE OF ***ALLERGIC*** ***RHINITIS*** PATIENTS TO SEGMENTAL ANTIGEN CHALLENGE CHARACTERIZATION OF EOSINOPHIL AND MAST CELL MEDIATORS.

AU SEDGWICK J B [Reprint author]; CALHOUN W J; GLEICH G J; KITA H; ABRAMS J S; SCHWARTZ L B; VOLOVITZ B; BEN-YAAKOV M; BUSSE W W

CS SECT ALLERGY/IMMUNOL, H6/367 CLINICAL SCI CENTER, 600 HIGHLAND AVE, MADISON, WIS 53792, USA

SO American Review of Respiratory Disease, (1991) Vol. 144, No. 6, pp.

1274-1281.
CODEN: ARDSBL. ISSN: 0003-0805.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 12 Feb 1992
Last Updated on STN: 13 Feb 1992
AB Segmental antigen bronchoprovocation was used to define the nature of the inflammatory process in allergic airway disease. Bronchoalveolar lavage fluid obtained from ***allergic*** ***rhinitis*** patients 12 min after segmental antigen instillation (immediate response) revealed a significant increase in histamine and tryptase, but no cellular response. Repeat segmental lavage 48 h later (late response) showed marked and significant increases in both low and normal density eosinophils as well as striking elevations of eosinophil granular protein levels (major basic protein, eosinophil-derived ***neurotoxin*** , eosinophil cationic protein, and eosinophil peroxidase). Leukotriene C4, but not tryptase, concentrations were also consistently elevated in late lavage samples. Further, the late lavage samples showed a significant increase in interleukin-5 concentrations that correlated with the presence of eosinophils and eosinophil granular proteins. Neither eosinophils nor soluble mediators of eosinophils increased when normal subjects were similarly challenged with antigen. These data suggest that eosinophils are attracted to the airway during the late-phase allergic reaction and that IL-5 may produce changes in airway eosinophil density and promote the release of granular proteins to cause airway injury.
TI IMMEDIATE AND LATE AIRWAY RESPONSE OF ***ALLERGIC*** ***RHINITIS*** PATIENTS TO SEGMENTAL ANTIGEN CHALLENGE CHARACTERIZATION OF EOSINOPHIL AND MAST CELL MEDIATORS.
AB. . . bronchoprovocation was used to define the nature of the inflammatory process in allergic airway disease. Bronchoalveolar lavage fluid obtained from ***allergic*** ***rhinitis*** patients 12 min after segmental antigen instillation (immediate response) revealed a significant increase in histamine and tryptase, but no cellular. . . both low and normal density eosinophils as well as striking elevations of eosinophil granular protein levels (major basic protein, eosinophil-derived ***neurotoxin*** , eosinophil cationic protein, and eosinophil peroxidase). Leukotriene C4, but not tryptase, concentrations were also consistently elevated in late lavage samples.. . .

L5 ANSWER 45 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 21
AN 1992:109335 BIOSIS <<LOGINID::20100927>>
DN PREV199242049335; BR42:49335
TI RAGWEED-SPECIFIC IGA IN NASAL LAVAGE FLUID OF RAGWEED-SENSITIVE ***ALLERGIC*** ***RHINITIS*** PATIENTS INCREASE DURING THE POLLEN SEASON.
AU REED C E [Reprint author]; BUBAK M; DUNNETTE S; BLOMGREN J; PFENNING M; WENTZ-MURTHA P; WALLEN N; KEATING M; GLEICH G J
CS MAYO CLIN AND FOUND, 200 FIRST ST SW, ROCHESTER, MINN 55905, USA
SO International Archives of Allergy and Applied Immunology, (1991) Vol. 94, No. 1-4, pp. 275-277.
Meeting Info.: 18TH SYMPOSIUM OF THE COLLEGIUM INTERNATIONALE ALLERGOLOGICUM ON CELLULAR AND MOLECULAR NETWORKS IN CLINICAL IMMUNOLOGY AND ALLERGY, FUNCHAL, MADEIRA, NORTH ATLANTIC OCEAN, SEPTEMBER 22-26, 1990. INT ARCH ALLERGY APPL IMMUNOL.
CODEN: IAAAAM. ISSN: 0020-5915.

DT Conference; (Meeting)
FS BR
LA ENGLISH
ED Entered STN: 24 Feb 1992
Last Updated on STN: 24 Feb 1992
TI RAGWEED-SPECIFIC IGA IN NASAL LAVAGE FLUID OF RAGWEED-SENSITIVE ***ALLERGIC*** ***RHINITIS*** PATIENTS INCREASE DURING THE POLLEN SEASON.
IT Miscellaneous Descriptors
IMMUNOGLOBULIN A EOSINOPHIL EOSINOPHIL-DERIVED ***NEUROTOXIN***

L5 ANSWER 46 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1991:220280 BIOSIS <<LOGINID::20100927>>
DN PREV199140106115; BR40:106115
TI THE IMPORTANCE OF NASAL ALLERGEN SPECIFIC IGA AND EOSINOPHILS IN SEASONAL ***ALLERGIC*** ***RHINITIS*** .
AU BUBAK M E [Reprint author]; WENTZ-MURTHA P E; DUNNETTE S L; KEATING M U; WALLEN N D; WEILER D A; BUTTERFIELD J H; BLOMGREN J A; PFENNING M A; ET AL
CS ROCHESTER, MINN, USA
SO Journal of Allergy and Clinical Immunology, (1991) Vol. 87, No. 1 PART 2, pp. 242.
Meeting Info.: FORTY-SEVENTH ANNUAL MEETING OF THE AMERICAN ACADEMY OF ALLERGY AND IMMUNOLOGY, SAN FRANCISCO, CALIFORNIA, USA, MARCH 1-6, 1991. J ALLERGY CLIN IMMUNOL.
CODEN: JACIBY. ISSN: 0091-6749.
DT Conference; (Meeting)
FS BR
LA ENGLISH
ED Entered STN: 5 May 1991
Last Updated on STN: 5 May 1991
TI THE IMPORTANCE OF NASAL ALLERGEN SPECIFIC IGA AND EOSINOPHILS IN SEASONAL ***ALLERGIC*** ***RHINITIS*** .
IT Miscellaneous Descriptors
ABSTRACT RAGWEED POLLEN IMMUNOGLOBULIN A IMMUNOGLOBULIN E EOSINOPHIL-DERIVED ***NEUROTOXIN***

L5 ANSWER 47 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
AN 1991:216363 BIOSIS <<LOGINID::20100927>>
DN PREV199140102198; BR40:102198
TI EOSINOPHIL DERIVED ***NEUROTOXIN*** EDN IN NASAL LAVAGES CORRELATES WITH WEEKLY HAY FEVER SYMPTOMS DURING SEASONAL EXPOSURE WHILE MAST CELL TRYPTASE DOES NOT.
AU BUBAK M E [Reprint author]; WENTZ-MURTHA P E; DUNNETTE S L; KEATING M U; WALLEN N D; WEILER D A; BUTTERFIELD J H; BLOMGREN J A; PFENNING M A; ET AL
CS ROCHESTER, MINN, USA
SO Annals of Allergy, (1991) Vol. 66, No. 1, pp. 66.
Meeting Info.: 47TH ANNUAL MEETING OF THE AMERICAN COLLEGE OF ALLERGY AND IMMUNOLOGY, SAN FRANCISCO, CALIFORNIA, USA, NOVEMBER 10-14, 1990. ANN ALLERGY.
CODEN: ANAEA3. ISSN: 0003-4738.
DT Conference; (Meeting)
FS BR
LA ENGLISH
ED Entered STN: 5 May 1991
Last Updated on STN: 5 May 1991

TI EOSINOPHIL DERIVED ***NEUROTOXIN*** EDN IN NASAL LAVAGES CORRELATES WITH WEEKLY HAY FEVER SYMPTOMS DURING SEASONAL EXPOSURE WHILE MAST CELL TRYPTASE DOES NOT.
IT Miscellaneous Descriptors
ABSTRACT HUMAN ANTIHISTAMINE THERAPY ***ALLERGIC*** ***RHINITIS***

L5 ANSWER 48 OF 48 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN DUPLICATE 22
AN 1989:518101 BIOSIS <<LOGINID::20100927>>
DN PREV198988134244; BA88:134244
TI MAJOR BASIC PROTEIN AND EOSINOPHIL-DERIVED ***NEUROTOXIN*** CONCENTRATIONS IN NASAL-LAVAGE FLUID AFTER ANTIGEN CHALLENGE EFFECT OF SYSTEMIC CORTICOSTEROIDS AND RELATIONSHIP TO EOSINOPHIL INFLUX.
AU BASCOM R [Reprint author]; PIPKORN U; PROUD D; DUNNETTE S; GLEICH G J; LICHTENSTEIN L M; NACLERIO R M
CS PROFESSIONAL OFFICE BUILD, SUITE 402, 5601 LOCH RAVEN BLVD, BALTIMORE, MD 21239, USA
SO Journal of Allergy and Clinical Immunology, (1989) Vol. 84, No. 3, pp. 338-346.
CODEN: JACIBY. ISSN: 0091-6749.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 15 Nov 1989
Last Updated on STN: 21 Nov 1989
AB The late-phase response to nasal challenge with antigen is associated with a mixed inflammatory cell influx in which the eosinophil demonstrates the earliest and greast proportionate rise. We investigated the evidence for activation of the esoinophil during the late response by measuring the concentration of the eosinophil-derived mediator major basic protein (MBP) and the eosinophil-derived ***neurotoxin*** (EDN) in nasal-lavage fluids before and for 11 hours after antigen challenge in 13 subjects withs easonal ***allergic*** ***rhinitis*** . The subjects received oral predinsone (20 mg three times daily) or placebo in a double-blind, crossover manner of 2 days before each of two antigen challenges. After placebo pretreatment, significant increases over diluent baseline (4.5 .+-. 0.4 ng/ml) occurred in the levels of MBP is nasal-lavage fluid during the early (9.8 .+-. 2.9 ng/ml; $p < 0.005$) and late (15.3 .+-. 4.8 ng/ml; $p < 0.01$) responses to antigen challenge. Significant increases ($p < 0.05$) in the concentration of EDN also occurred during the late response to antigen that correlated with the levels of MBP ($r = 0.48$; $p < 0.001$). The cumulative late-phase increase in MBP correlated closely ($rs = 0.96$; $p < 0.005$) with the total influx of eosinophils. Oral prednisone pretreatment significantly reduced the mean of each subject's peak late-phase concentration of both MBP (30.7 .+-. 5.8 ng/ml versus 13.3 .+-. 4.3 ng/ml; $p = 0.005$) and EDN (885 .+-. 659 ng/ml versus 71 .+-. 41 ng/ml; $p < 0.05$). These data provide evidence for eosinphil degranulation during the late response and inhibition of this response by prednisone, supporting its pathogenetic role.
TI MAJOR BASIC PROTEIN AND EOSINOPHIL-DERIVED ***NEUROTOXIN*** CONCENTRATIONS IN NASAL-LAVAGE FLUID AFTER ANTIGEN CHALLENGE EFFECT OF SYSTEMIC CORTICOSTEROIDS AND RELATIONSHIP TO EOSINOPHIL INFLUX.
AB. . . esoinophil during the late response by measuring the concentration of the eosinophil-derived mediator major basic protein (MBP) and the eosinophil-derived ***neurotoxin*** (EDN) in nasal-lavage fluids before and for 11 hours after antigen challenge in 13 subjects withs

easonal ***allergic*** ***rhinitis*** . The subjects received oral predinsone (20 mg three times daily) or placebo in a double-blind, crossover manner of 2 days. . .

IT Miscellaneous Descriptors

HUMAN PREDNISONE IMMUNOSUPPRESSANT-DRUG DEGRANULATION LATE-PHASE RESPONSE SEASONAL ***ALLERGIC*** ***RHINITIS***